

OBSERVATIONS ON SOME CAUSES
OF SUDDEN CARDIAC DEATH

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SECTION I

INTRODUCTION

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INTRODUCTION

Sudden death has been recorded in the literature from time immemorial. Many of the causes were attributed by early workers to over eating, severe emotion and even a visitation from God. The best known work in those early times was that of Giovanni Maria Lancisi (1654 - 1720) who, at the request of Pope Clement XI, in 1705, investigated the causes of sudden death occurring in Rome. This work was published in 1707 under the title of De subitaneis mortis (on sudden death). Thereafter many workers investigated this subject. Giovanni Battista Morgagni (1682 - 1771) described in detail two cases of sudden death occurring in young individuals. One case began, "A young man, of about twenty-seven years of age, had been afflicted, already, for had been often seiz'd with a shortness of breath; which, however, in a short time after a vein was open'd, remitted. One day, as he was at his devotions, he fell down suddenly; his face was pale, he could scarcely speak; he soon died. In the thorax was seen a large aneurism. For when the carotid arteries came off from the aorta, it began, and extending itself to the sternum, to which it then strongly

adhered /

adhered, so that it could not be separated without laceration. It was also produc'd under the right clavicle to the third and fourth rib, the internal surface of which had hollow'd out and then render'd rough and unequal. Finally, it came within the pericardium where, being ruptur'd, it had fill'd all the cavity of it with blood. There was no polypous concretion of the heart". The other; "A strumpet of eight-and-twenty years of age, of a lean habit, having complain'd for some months, and particularly for the last fifteen days, of a certain lassitude, and a loathing of food, and almost of everything, for this reason made less use of other aliments and more of unmixed wine; to the use of which she had always been too much addicted. A certain debauchee having gone into the house to her, and after a little time having come out, with a confus'd and disturbed countenance and she not having appear'd for two or three hours after, the neighbours, who had observed these things, entering in, found her not only dead but cold; lying in bed with such a posture of the body, that it could not be doubted what business she had been about when she died, especially as the semen verile was seen to have flow'd down from the organs of generation. I was, therefore, ask'd whether I desired to have the genitals or not? and whether I would have the other viscera also? I answered that I should be glad of both; not that I expected to see

anything /

anything particular in the organs of generation, now that the semen have flow'd out, but that I wish'd to take fresh notice of some things which I had often observ'd; for I conjectur'd, as I then said, that the cause of this sudden death would certainly be found to consist in the rupture of some large vessel. It was at this time extremely hot, it being June of the year 1725, nor were we allowed to dissect the body till about the later end of the following day. The neck was livid under the chin, yet without any marks of force having been externally applied. The back was also somewhat livid. The abdomen was tense and did not shew any mark of the woman's having ever been pregnant. The uterus being taken away, the small intestine appear'd very red. The large intestines, and especially the lower ones, were full of excrements; the stomach was very large although almost empty. There was serum extravasated into the belly, to about the quantity of a pint, not unlike turbid water in which fresh meat had been wash'd, and so acrid, that it effected the extremities of the fingers with the sense of a kind of heat. In the thorax, the lungs were so far off their natural colour, that they were not black even on the posterior part. But the pericardium was so distended, that no sooner was a little wound made into it, but a serum burst forth, of the same nature with that which had been

seen /

seen in the belly. Yet still a great quantity remain'd, and under it a black and firmly concentrated blood cover'd the surface of the heart. Which being brought to me on the following day, in the morning, together with the large vessels and the genitals, I observ'd, before I cut into them, that neither the heart itself, nor the trunk of the great artery, was dilated, and even that both of them, by their smallness, corresponded very well with the stature of the woman, which they said had been rather small. Being then about to lay open that artery from the inferior extremity, which was at the septum transversum, and having seen that one side of it, not much higher, was black to the extent of five or six fingers breath; I found that this was owing merely to the effusion of blood into the cells of the external coat; for other parts were quite in their natural state. But an internal disease began from the left extremity of the curvature of the aorta, and going from thence quite to the heart, became so much the larger in proportion as the artery came nearer to the heart. That is to say, whitish marks of a future ossification occur'd; in others; some small foramina, as it were, had begun to be form'd, and in still other places were parallel furrows, drawn longitudinally; and in this manner was the surface of the artery unequal here and there. But when I came near to the

semilunar /

semilunar valves, which seem'd to be lank and contracted, at the distance of half an inch above that which lies on the back-part, was an orifice that would have admitted the end of a man's thumb by means of which the aorta communicated with a roundish aneurism, that hung to it in the form of a sacculus. This sacculus exceeded the size of a walnut before it is stripp'd of its green coat; and was so plac'd in the back of the aorta, that, as it inclin'd a little to the left side, it seem'd that it could not happen otherwise, but it must have obstructed the offices of the left auricle, or the adjoining sinus. And it had been ruptur'd in the upper part by the blood flowing from thence into the pericardium through a small foramen, the edges of which were lacerated and black. The internal surface of the sacculus was invested with red and polypous pellicles, which, like the texture of the onion, you might divide into as many strata or lamina as you pleas'd. But in the auricles and the ventricles of the heart, neither was there any polypous concretion, nor did the least quantity of blood remain."

Hope, in 1831, discussed sudden death and described two causes for this, namely, "(1) Softening of the heart with increased intensity of redness - namely claret or violet coloured, denoting an excess of blood in the muscular substance and, (2) softening with diminution of redness, namely faint yellow or

fawn /

fawn coloured and bespeaking a deficiency of blood".

Stokes, in 1854, enumerated the following conditions as causes of sudden death:-

- (1) Abscess in the wall of the heart.
- (2) Apoplectic effusions into the substance of the heart, as described by Cruveilhier in his "Anatomie Pathologique du corps humain."
- (3) Muscular aneurysm.
- (4) Fatty degeneration.

The majority of these descriptions is eventually associated with some form of disease of the heart or great vessels, although it is understood that other conditions may bring about sudden death.

C A S E M A T E R I A L

In this thesis it is intended to discuss the aetiology of sudden cardiac death from natural causes where no antecedent disease was suspected during life.

This is drawn from an analysis of my own records of necropsies for the Medico-Legal Department of the Ministry of Justice of Egypt between November, 1938 and November, 1947.

Necropsies were conducted in different centres of the Department. The work was done in the principal centres and the general Mortuary in Cairo from November, 1938 to December, 1941; the Assuit centre including the Provinces of Souhag, Qena and

Aswan from January, 1942 to June, 1944; again in Cairo from July, 1944 to September, 1945; in Alexandria from October 1945 to March 1946; again in Cairo from April 1946 to November, 1947; then for one and a half months each year in the summer of 1945, 1946 and 1947 when I was stationed on temporary relief in Zagazig which is the capital of a Province in the Delta; in Beni Sweif capital town of the second southern Province of Upper Egypt, and also in Minya the capital town of the third southern Province.

In the capitals and towns necropsies were performed in Government General hospitals, Fever hospitals Chest hospitals, Maternity hospitals, Children's hospitals and private hospitals. In the villages these were done at homes.

The autopsies also cover cases which died in asylums, prison hospitals of the principal towns and prison cases which died actually in the cells. Last form a group of cases which frequently showed no signs of morbidity to warrant admission to the prison hospital.

There remains a group of cases which were found dead on the road and others which died suddenly in offices, public places or at home.

It is necessary to point out that these are not the ordinary cases seen in every day pathological practice in the big towns, where statistics are not

likely /

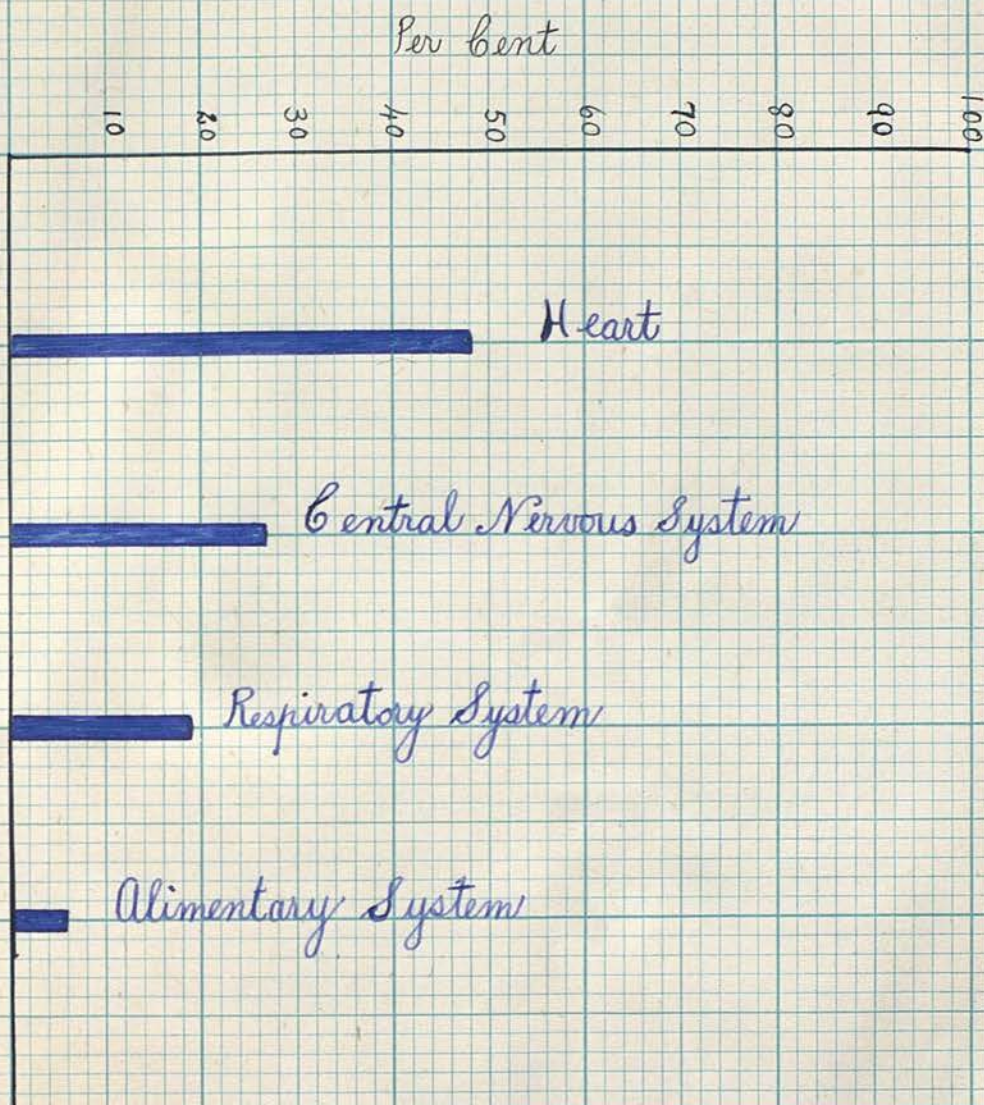


Fig. 1
Sudden Death

likely to differ much from one hospital to another, but represent collectively sudden cardiac death in widely different parts of the country from the biggest city to the smallest country village.

Autopsies including all forms of death, ranging from simple natural death to violent murder were 3,250. These covered all ages from birth to age of 90.

Death from all natural causes occurred in 1,980 cases - 60.9%. Death was sudden in 760 cases accounting for 38.4%. The latter are grouped according to the systems involved and expressed per cent as indicated in Fig. 1.

The time interval between the occurrence of the fatal symptoms and death was fixed at approximately six hours in the various groups of Fig 1. This was made use of for three main purposes:-

1. Reasonable comparison of the incidence of sudden death in the different systems of the body.

2. Avoiding including autopsies performed after the process of putrefaction has set in, where a pathological diagnosis could not be adequately ascertained in a hot country like Egypt, and especially in the summer time when humidity is high in many parts and the putrefactive process in the corpses would be rapid and progressive.

3. Keeping within the limit of the term "sudden" as much as possible.

S U D D E N D E A T H

It is necessary before proceeding further to define sudden death. One should class as "sudden" any death which was unexpected and in which the process of dying was rapid.

Death occurring dramatically within seconds or minutes is classed as "instantaneous".

It may be helpful to attempt an explanation of sudden death rather than define it dogmatically. Death is considered sudden if it is not delayed more than a few hours after the onset of the first fatal symptoms. The time factor is important, its duration is not a fixed matter to decide upon rigidly, but it must fall within a reasonable limit. In that respect six hours would represent the upper limit. Death occurring more than six hours after the onset of symptoms is a protracted one in which death can be expected at any moment.

The following views were expressed by other observers:- Brouardel (1902) described sudden death as the rapid and unseen termination of an acute or chronic disease which has in most cases developed in a latent manner. Hamman (1934) explained sudden death as one coming quickly and unexpectedly. Lisa (1939) considered death as sudden when occurring instantaneously or within a few minutes (10-20) after collapse. Weiss (1940) expressed /

expressed sudden death as an unexpected death occurring within several minutes or even hours after the onset of alarming symptoms. Burch and Winsor (1942) stated that death is sudden if it occurs within six hours from symptoms. Smith (1943) considered "sudden" as unexpected and dramatic. Boyd (1944) defined "sudden" as either occurring instantaneously or in the course of a few minutes or hours. Glaister (1945) referred to "sudden" when termination of life comes quickly under circumstances when its immediate arrival is unexpected. Simpson (1946) defined "sudden" as occurring unexpectedly especially when the possibility of disease seems remote. He again, in (1947), referred to it as unexpected, precipitating death within a few hours of apparently good health. He also included "instantaneous" under sudden death. Kerr (1946) considered sudden death as one preceded or only preceded for a short time by morbid symptoms. Deadman (1947) included under "sudden", instantaneous death as occurring in a matter of seconds or minutes and unexpected death which may take hours or days. Emara and Soliman (1948) stated that by sudden death is meant unexpected death occurring under suspicious circumstances from apparently unknown causes, or following a brief or obscure illness.

It is, therefore, evident that the definition of sudden death described is largely in accordance with others.

THE HEART IS THE VICTIM OF DEATH

In the struggle between life and death the heart seems to stand alone. This appears at a glance from fig. 1, which shows the higher incidence of cardiac death.

From this fact one should realise that there is no place for an element of surprise when one reflects why nearly half the persons in an apparently normal state of health should succumb to sudden cardiac death. The high proportion of cardiac death may point to the importance of this organ in the human machine as it is the one which is selectively struck by the insult of death.

It may be argued that the heart is the least resisting and self supplying organ, but if that is so, is it because of a natural structural delicacy or an unexplained sensitivity to many diseases, if not the most of them?; or is it because fellow organs do not support the heart in the battle of death? If such is the case, then the heart has been defied by those whom it has supported all its life.

But after all, death knows what to strike; it strikes primarily the most vital centre; the heart, just as one shakes a tree to obtain its fruit.

That cardiac death is responsible for a great proportion of sudden death is evident from the

following /

following statistics in the table set out below:-

Sudden Cardiac Death

AUTHOR	Year	Per cent of All Sudden Deaths
Gonazles et al	1940	42.0
Burch and Winsor	1942	78.0
Moritz and Zamcheck	1946	46.9 (average)
Kerr	1946	65.0
Simpson	1947	57.5
Deadman	1947	65.0
Present series	1949	48.0

The Public Health Report (1944) on the morbidity and mortality from specific causes during the year 1943 and the recent preceding years in the various states and districts of Columbia, stated that the crude death rate for heart disease showed an increase of 4.4% in the year 1943 as compared to 1940.

In this connection, it is interesting to note that this increase in 1943 occurred during the height of the war and that such a relation may suggest an aetiological factor. However, in the present series this was not observed.

S E L E C T I O N O F C A S E S

Cases under study exclusively represent disease
process /

process of the heart and its direct tributaries. To keep within the sense of the word "cardiac", the abdominal aorta and pulmonary infarctions were not included. Cardiac death, under anaesthesia, was also excluded, because in these cases a morbid condition has already been present.

Thus keeping to the definition of "sudden" and adhering to the word "cardiac", case material was selected.

H I S T O R Y O F C A S E S

Wherever it was possible, histories were obtained.

Histories of patients who died in hospitals were either provided by the patients themselves or by relatives. In those who died at their homes the history was obtained from relatives and in those found dead on the road, the history was available in a number of cases.

Unknown persons found dead on the road were either identified later, or remained unknown. However, burial was delayed for about three days to search for identification in those cases received at the general mortuary in Cairo and in Alexandria. Unfortunately a reliable history was wanting in them all. However, the proportion of these cases was

negligible./

negligible.

It is the practice of the pathologist in all cases to obtain the history by himself from relatives or friends of the deceased besides that given by members of the police or parquets, as the latter, in a number of cases, may be unsatisfactory from a medical point of view. Unfortunately, in a number of cases, the history obtained, in either way, was irrelevant owing to the ignorance of relatives in a proportion of cases, or because they volunteered to give an imaginary satisfactory account in the hope that an autopsy could be suspended. These have been discarded.

Cases which were not autopsied at once by members of the Medico Legal Department were examined by the local Medical Officer of Health, whose duty it was to report on the external examination of the body and ascertain the time of death.

PRESENTATION OF THE CASES

The scheme adopted is presented in two sections, Section II and Section III.

1. Section II.

A. In this section six chapters are included, each dealing with a cause of sudden cardiac death.

B./

B. The cases in each chapter were grouped on a pathological basis alone.

C. The aetiology of cardiac death in these groups of cases was drawn from consideration of:-

- (i) Incidence of the disease.
- (ii) Age incidence.
- (iii) Sex incidence.
- (iv) Race.
- (v) Body constitution.
- (vi) Past history.
- (vii) Social conditions.
- (viii) Associated diseases.
- (ix) Predisposing factors.
- (x) Fatal manifestations and their duration.

Although the latter are not aetiological factors of significance, they are of importance. Not many cases of sudden death are witnessed by practitioners at this stage and it may be useful to be acquainted with warning symptoms, although in a number of cases these are minimal or even absent.

D. In each group a comparative study was made from observations in literature. The literature mainly consulted included the last fifteen years which expressed up to date views. However, on many occasions, older literature was also consulted, whenever the necessity for this arose.

E. A historical review preceded all the
 chapters /

chapters. In this connection it may be interesting to note that present day views on some diseases have not advanced with the march of time.

2. Section III.

Conclusions were drawn from observations made from the cases, and these collectively were inserted in this section.

3. References.

References were given in alphabetical order after the last section.

SECTION II

COMMON CAUSES

OF

SUDDEN CARDIAC DEATH

(4) Aortic dissection	2	0	2	0.6
(5) Aortic aneurysm	5	0	5	1.4
(6) Atherosclerosis of the heart	2	0	2	0.6
(7) Hypertrophy of the heart	1	0	1	0.3
(8) Myocarditis	10	14	24	6.7
(9) Valvular diseases	14	3	17	4.7
(10) Rupture of aneurysm of the aorta	0	0	0	0.0

Diagnosis of these causes, as grouped above,

is carried out in the next six chapters.

S E C T I O N I I

COMMON CAUSES OF SUDDEN CARDIAC DEATH

On a pathological basis the following diseases were recognised causes of sudden cardiac death. These are tabulated in some detail below.

Common Causes of Sudden Cardiac Death

CAUSES	MALES	FEMALES	TOTAL	PER CENT
<u>I. Coronary Artery Disease</u>				
(a) Coronary Sclerosis	160	52	212	58.0
(b) Acute Coronary Occlusion	60	25	85	23.1
(c) Coronary Ostial Occlusion	2	1	3	0.9
<u>II. Spontaneous Cardiac Rupture.</u>				
(a) Acute Infarction	2	0	2	0.6
(b) Cardiac Aneurysm	5	0	5	1.4
(c) Adiposity of the heart	2	0	2	0.6
<u>III. Adiposity of the heart</u>	1	3	4	1.1
<u>IV. Myocarditis</u>	18	14	32	8.7
<u>V. Valvular Diseases</u>	14	3	17	4.7
<u>VI. Rupture of Aneurysm of the Aorta</u>	3	0	3	0.9

Discussion of these causes, as grouped above, is carried out in the next six chapters.

CHAPTER I

CORONARY ARTERY DISEASE

CORONARY ARTERY DISEASE

Historical Review:

The name coronary arteries of the heart, *arteria coronaria cordia*, was first used by Galen (A.D.130-200). In spite of the use of the term cardiac arteries, *arteria cardica*, by Winslow in 1732, the Galenic name has persisted. The earliest accurate description of the course and branches of the coronaries was given by Vieussens in 1715. Lower, in 1728, inferred that there is a free anastomosis between one coronary artery and the other (Cowdry, 1933, p. 431). However, illustration of the coronary arteries appeared as early as 1543 in the *De Fabrica* of Vesalius (Herrick 1942, p. 208).

Our present knowledge of arteriosclerosis had its origin in the relatively crude observations of the anatomists of the sixteenth century. Written descriptions of arteriosclerosis in man first appeared in the works of the anatomists who followed Vesalius. They looked upon the condition as a natural phenomenon related to the advance of life, and their discussions centred chiefly around the presence or absence of, "bone in the heart", a condition described by Aristotle and his predecessors in certain animals (Cowdry, 1933, p. 19).

By the year 1600 all educated physicians were
probably /

probably aware of the condition of "ossification" of the arteries, particularly the aorta. No outstanding advances in the understanding of arteriosclerosis were made in the seventeenth century. William Harvey (1578 - 1657), Franciscus de le Boë Sylvius (1614-72), Thomas Bartholin (1616-80), Thomas Willis (1621-75) and Marcello Malpighi (1628-94), mentioned arteriosclerosis but no one seems to have thought the subject worthy of investigation.

William Cowper (1666-1709) called attention to the fact that in the thickened calcareous arteries the passage of blood was impeded.

The early eighteenth century was notable for the appearance of a number of important treatises on the physiology and pathology of the vascular system.

Johann Friedrich Crell (1740) dealt with hardening of the coronary arteries, and explained its basis on a tophaceous nature derived from pus, (i.e. the atheromatous matters), the inner part of which had dissipated while the thicker part had hardened. He made the further point that induration was not confined to senility, but might occur at any part of life whatsoever.

Giovanni Battista Morgagni, in his treatise, "On the Seats and Causes of Disease", 1761, gave minute descriptions, clinical correlation and exposition of the pathological lesions encountered in

hardening /

hardening of the arteries. He also noted the increased size of the heart in some cases with extensive hardening of the arteries, and recorded the association of pain in the chest and "ossification" of the coronary arteries. He pointed out that the arterial changes were not always found in senility, citing a case of a woman of ninety in whom the lesions were minimal (Cowdry 1933, p.28).

In 1772, William Heberden had named and given his classical account of angina Pectoris. He had seen some twenty cases but had not yet studied a single case postmortem (Segall 1945). In his dissertation entitled "Some Account of a Disorder of the Breast", presented to the College of Physicians of London on July 21, 1768, he did not describe the story of any individual patient, but presented the clinical syndrome as a whole. Its publication in the Medical Transactions of the College of Physicians in 1772, had initiated an anonymous physician, who wrote to Heberden, (1785), describing his own symptoms which were those of angina pectoris. He closed the letter with the following sentence, "But, be the cause what it may, if it please God to take me away suddenly, I have left direction in my will to send an account of my death to you with permission for you to order such examination of my body as will show the cause of it, and, perhaps tend at the same time to be a discovery of the origin of that disorder, which is

the /

the subject of this letter, and be productive of means to counteract and remove it". Heberden's publication of the letter and the autopsy of the anonymous physician's death in the third volume of the Medical Transactions of the College of Physicians (1785) constitutes the first clinico-pathological report of a single case of angina pectoris.

John Fothergill, in 1776, reported the association of precordial pain with changes in the state of the coronary arteries. Before Fothergill, however, Edward Jenner had communicated his own similar views based on his reading and necropsy observations to Heberden. He also reported these privately at a meeting of a London Medical Society, but refrained from publishing them at the same time out of solicitude for his teacher, John Hunter, who was a victim of the malady. After Hunter's death in 1793 he turned over his material to Caleb Parry (1799) of Bath, who published it in his famous, "Inquiry into the Symptoms and Causes of Syncope Anginosa".

The nineteenth century inaugurated a new era in general pathology credited to the French, English and Vienna Schools, though arteriosclerosis received attention from all.

Xavier Bichart (1801), located the initial lesions of arterial "ossification" in the intima, reaching this decision by careful dissection.

Joseph Hodgson in 1815 considered atheromatous
changes /

changes the result of arterial inflammation and the contents of the soft lesions themselves, true pus. He compared the sloughing of atheromatous spots to ulcers of the small bowel, ("these elevations had sloughed out in the same manner as the glandular aggregatae of the intestines become converted into cheesy matter and slough"), noting the disappearance of the curdy matter in the blood stream. Considering the occurrence of true bone formation in the calcareous plates of arteries, Hodgson, upon a chemical analysis taken up by his friend, Mr. Brande, was convinced that it was an earthly deposit and not true bone.

Jean-Frederic-Martin Lobestein opposed the idea, already generally accepted, that angina pectoris was a result of coronary artery disease, noting its occurrence in absence of this lesion and vice versa, (Cowdry 1933, p. 32-36.).

Hope (1831) described lesions of the heart and the coronary arteries, in relation to angina pectoris, in the order of osseous, cartilagenous, steatomatous and degenerative. This relation was also recognised by Stokes (1854), giving "disease of the aorta, with or without obstruction of the coronary arteries", among other causes of angina pectoris.

The first case of coronary occlusion correctly diagnosed during life was communicated by Adam Hammer,

"On /

"On May 4, 1876 at 9 a.m. my young friend and colleague, Dr. Wichmann, took me with him to see a patient, whom, as he remarked, he did not understand, -----.

Status prasens: I found the patient in the following condition: body stretched out in bed, slightly elevated, his hands resting on the covers. Pulse, 8 beats to the minute (half as frequent as on the preceding evening at 10 o'clock) at regular intervals, that is, one contraction of the heart every 8 seconds. Face and skin of the entire body pale, cool and covered with sticky sweat. The eyes clear, pupils of moderate size, reacting easily, around the lips a light tinge of cyanotic coloring, tongue and mucous membranes of the mouth and throat pale and anaemic, no dyspnoea, no cough, no expectoration and no pain in any place. Respiration 24 to the minute. Sensorium quite free. The patient produces the impression, by his bearing, the expression of his countenance and conversation, that he has no idea of the seriousness of his malady. ----- The heart beat weak, one every 8 seconds. Upon the sound of each systole and diastole which although weak could be clearly made out and were without any murmur, there followed immediately a clonic spasm of the heart, which beat forcibly upon the applied ear with a sort of rustling, lasted exactly 5 seconds with the same intensity and then ceased as if it cut off.

I can compare these rapid successive twitchings of the heart muscle with nothing better than with the marked tremor of the hand of a man who is suffering from delerium tremens ----- I thought that only a sudden, progressively increasing disturbance in the nutrition of the heart itself such as a cutting off of the supply of nourishment could produce such changes as this case showed, and that such an obstruction could be produced only by a thrombotic occlusion of at least one of the coronary arteries. From lack of ground for any other satisfactory explanation, I was carried away by this thought. I mentioned my conviction to my colleague at the bed side. He, however, had a non-plussed expression and burst out, "I have never heard of such a diagnosis in my whole life", and I answered, "Nor I also".----- Remarkable to relate the patient lived 19 hours longer, he died early on May 5. ----- On May 6 (29 hours after death) at 11 a.m. we carried out a partial autopsy, while the body was already in its coffin----- I split the aorta and extended the incision through the commissure of the posterior and right valves, which proved to be very useful.----- What first and principally struck one's view was the marked tension and stretching of the right valve by a mass which not only filled the right sinus of valsalva but also bulged out like a half sphere. On removing this

mass /

mass carefully and examining it carefully, it was found that the upper layers from above to the origin of the coronary arteries inside the sinus were composed of fresh, coagulated, jelly-like, whitish-yellow material mixed with blood --- On the right and left valves there were fresh white soft endocarditic excrescences" (Major 1945).

Herrick, gave an account of his first case of coronary occlusion of a man, aged 55 years, who on January 16th, 1910, was seized, an hour after a moderately full midnight meal, with severe pain in the lower precordial region, and died suddenly 52 hours after onset of pain. In autopsy, he described a myocardial infarction result of an occlusion of the left coronary artery.

Later, in 1912, when he read before the Association of American Physicians his first paper on "Coronary Occlusion", which seemed to him to contain an important announcement, it fell like a dud, as no one except Emanuel Libman discussed it or even asked a question.

A vivid description of what might have been an early case of coronary thrombosis which antedates considerably any others reported is given below, (Stoll, 1934).

"G.B., aged eighty years, an itinerant teacher of philosophy, immediately after eating a heavy meal was seized with severe pain and great prostration

and /

and died a few hours later. The facts in the case are as follows:

A feast has been provided by the blacksmith of a small village. The menu was diversified and abundant. One dish was especially pleasing to the guest of honour, and he ordered that no one else should be served this particular food. There is some doubt as to just what this was but it seems probable that it consisted of pork. At any rate, after eating all he could, he evidently felt some discomfort, as he ordered that the remaining food be buried in a hole in the ground, remarking that no one else would be able to digest it. Almost immediately thereafter he was taken violently ill and suffered extreme pain. He is said to have passed some blood, probably from the bowels. He tried to conceal his suffering so as not to interfere with the festivity of the occasion and started forth on a journey on foot with some friends but very soon had to stop because of weakness and pain. He sat down on a blanket and evidently suffered from shock as he complained bitterly of thirst, asking for water three times before it was possible to procure any. Feeling somewhat better, he proceeded on his way, but the increasing pain again forced him to stop, and this time he was evidently so weak to sit, as he lay down. It is stated that he lay on his right side.

With /

With the great determination that had always characterised his life, he again attempted to proceed but was forced to lie down again; as formerly he lay on his right side. The pain was obviously much less after he ceased the exertion of walking as he was able to converse with friends at considerable length. As his illness came on at once after dining, quite naturally some one suggested that it was due to something he had eaten. Accordingly, he sent for his host and publicly exonerated him for any responsibility in connection with his illness. This gracious act appears to have been nearer the truth than the generous sufferer may have suspected. He did not succumb until a number of hours after the onset of the attack.

The old gentleman whose final illness has been described, died in the fifth century B.C. His name was Gotama Buddha.

ATHEROSCLEROSIS IN ANCIENT EGYPT

Ruffer (1911), who was associated with the Cairo Medical School, has proved, in his pathological studies, that atherosclerosis was a very common finding in ancient Egyptian mummies. He stated that the wonderful and amazing embalming process which was known in Egypt in those very far ages, and
which /

which is still a hidden secret up to the present date in spite of the development of science and, no doubt, the favourable influence of a dry climate have preserved hundreds of bodies in a state suitable for pathological examination.

Interestingly enough, first notice of this condition attended the opening of the body of one of the most widely known of all ancient Royal Egyptians, Menophthah, the reputed Pharaoh of the Hebrew exodus, who seems not to have died in the Red Sea as conventionally taught. This body, found in the tomb of Amenhotep II, was unwrapped by G. Elliot Smith, who sent a piece of the aorta to S.G. Shattock in London, where it was exhibited before the Royal Society of Medicine. Shattock (1903) was able to secure good microscopical sections, which revealed typical advanced sclerosis with extensive deposits of calcium phosphate.

Ruffer, in his studies, separated the mummies in chronological origin, the earliest being inherited from the XVIIIth Dynasty and the last from the XXVIIth, a range of about 2,000 years (approximately 1580 B.C. to 525 A.D.). The soft tissues of the mummies were soaked in a weak solution of sodium carbonate and formol, and thus the blood vessels were removed without tearing.

Atherosclerosis and calcification of vessels were striking findings in old and young individuals, although /

although a fair proportion of normal vessels was encountered in both ages. Even atheromatous ulcers were identified.

Monckéberg sclerosis was among other findings.

The conclusion of Ruffer was that atherosclerosis was at least as common among the ancient Egyptians and their neighbours (Greeks were included in his series) as it is today, and perhaps even exceeded the modern incidence.

It is noteworthy, in this connection, to state that in no instance had Ruffer found arterial lesions suggesting syphilitic infection. This agrees with the bulk of scientific opinion, that syphilis did not occur in ancient Egypt, (Cowdry 1933).

AN ANCIENT EGYPTIAN PAPYRUS

Did ancient Egypt know of the heart and circulation? The clue lies in the discovery of the Ebers Papyrus (2000 to 1500 B.C.) at Thebes (the capital of ancient Egypt) in 1862 by Edwin Smith, his daughter, the New York Historical Society, Dr. Breasted and the Oriental Institute.

The document, (Hamburger 1939), a surgical treatise, is of the highest importance to science. The papyrus is now known as "Edwin Smith Surgical Papyrus".

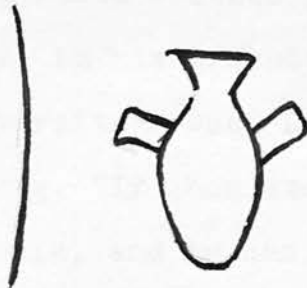
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That Dr. Breasted was aware of the significance and interest of the papyrus to surgeons, physicians, and physiologists is shown in his dedication, "To the memory of William Harvey, discoverer of the circulation of the blood, at the three hundredth anniversary of his great discovery, this publication of the earliest known surgical treatise is dedicated".

The treatise contained a systematic description by a surgeon, (Imhotep?), of 48 cases of injury and disease with notes of the examination, diagnosis, prognosis and treatment. In the introduction (page 16), Breasted, in discussing knowledge possessed by that surgeon writes, "He knew of a cardiac system and was surprisingly near recognition of the circulation of the blood, for he was aware that the heart was the centre and pumping force of a system of distributing vessels. He was already conscious of the importance of the pulse and had probably already begun to count the pulse,----". On page 13, in the general introduction, Breasted states, "--- there is much probability that the surgeon counts the strokes of the pulse, and it is doubtless a significant fact that the first physician who is known to have counted the pulse, Herophilus of Alexandria, (born 300 B.C.), lived in Egypt ----- Herophilus used an Egyptian water-clock for timing the count of the pulse". Breasted continues on page 64, "In observing the pulse /

pulse, our surgeon ----- states that observation of the pulse is undertaken 'in order to know the action of the heart' ". In an examination by the surgeon of a brain injury, "he probes with his fingers to the interior and discovers cardiac pulsations, or as he says 'fluttering and throbbing like that on the crown of an infant's head before it has grown together' ."

The following is a copy of the hieroglyphic analogue of the Egyptian word for "heart" taken from line 7, column 1, of the Edwin Smith Surgical Papyrus". University of Chicago Press, Chicago 1930 (Hamburger).



The hieroglyphic heart sign or word identifies the base and apex of the heart and the bilateral symmetrical projections, the pulmonary artery and aorta.

The first reference to the heart and circulation (pulse) and methods of their examination are found in Case 1 of column 1, which Dr. Breasted translates as follows. "Now if the priest of Sekhmet or any physician put his hands (or) his fingers (upon the head, upon the back of the) head, upon the two hands, upon the pulse, upon the two feet (he) measures (h'y) (to) the heart, because its vessels are in the back of

the /

the head and in the pulse; and because its (pulsation is in) every vessel of every member". The words enclosed in brackets () represent those supplied by the translator, because of the "broken and fragmentary" condition of this portion of the column.

A second reference to the heart appears in Case VII, column III, line 3, of the treatise "his heart ('his spirt' or 'his mind') is too weary to speak, or 'for speech'. His heart beats feebly". The phrase "too weary to speak" reflects the skill and artistry of the ancients.

The following still arouses general surprise and wonder. B. Ebbel, in his translation of the Papyrus Ebers (Oxford University Press, London 1937), disclosed the following, "If thou examinest a man for illness in his cardia, and he has pains in his arms, in his breast (mamma) and in one side of his cardia, and it is said of him: it is W 3 d - illness, then thou shalt say thereof = it is (due to) something entering the mouth, it is death that threatens him".

Could that be a description of angina pectoris?

-----oOo-----

C O R O N A R Y S C L E R O S I S

Case Material:

Coronary atherosclerosis, or coronary sclerosis, was encountered in 212 cases; an incidence of 58%. In all cases under study there was a severe involvement of the coronary arteries, and death was considered as being due primarily to coronary disease. None of the patients was hospitalised.

Classification of the Cases:

In this particular group of cases, in addition to the general pathological classification already referred to, it was found that two major groups emerged and presented two definite pathological entities. These groups were:-

- (1) Coronary sclerosis with cardiac hypertrophy.
- (2) Coronary sclerosis without cardiac hypertrophy.

Cardiac hypertrophy was considered indicative of a coexisting arterial hypertension. Accordingly, further classification adopted was as follows:-

- (1) Coronary sclerosis with hypertension.
- (2) Coronary sclerosis without hypertension.

The criteria used in classifying the hypertensive
group /

group were threefold:-

- (i) Evidence of Cardiac hypertrophy as stated above.
- (ii) Absence of valvular lesions which may account for hypertrophy.
- (iii) Absence of any other cause of cardiac hypertrophy.

Heart weights recorded in this series as indicative of hypertension were 450gm. or more in males and 400gm. or more in females.

According to this classification there were:-

- (a) 98 cases in the hypertensive group.
- (b) 114 cases in the non-hypertensive group.

The ratio of the non-hypertensive group to the hypertensive group was about 1.2 : 1 respectively.

I. Age Incidence:

In general, a wide range of age incidence of death occurred. The youngest person was 25 and the oldest 70 years of age. Death at both these two extremes of age occurred in males. No deaths occurred in females before the age of 40 years. The ages of death of the youngest and oldest hypertensive and non-hypertensive males and females are given in the following table:-

Table overleaf /

Sex	Youngest Hypertensive	Oldest Hypertensive
M	35	60
F	40	55
	Youngest Non-hypertensive	Oldest Non-hypertensive
M	25	70
F	40	60

This range of age in the two groups shows that:-

(1) In males

- (a) In the hypertensive group death occurred later in the younger age group, and earlier in the older age group.
- (b) In the non-hypertensive group death occurred earlier in the younger age group, and later in the older age group.

(2) In females

- In both hypertensive and non-hypertensive groups death before middle life occurred at the same age group (40 years).
- (3) In both sexes the span of life is shorter in the hypertensive group.
- (4) In both groups the span of life was shorter in the female than in the male sex.

Age incidence is also plotted graphically in
figs: 2, 3, 4, and 5.

Fig. 2. shows age incidence of all cases at death.

- (a) The highest incidence of death occurred at age of 50 years (45.8%).
- (b) The next highest incidence occurred at age of 45 years (18.9%).

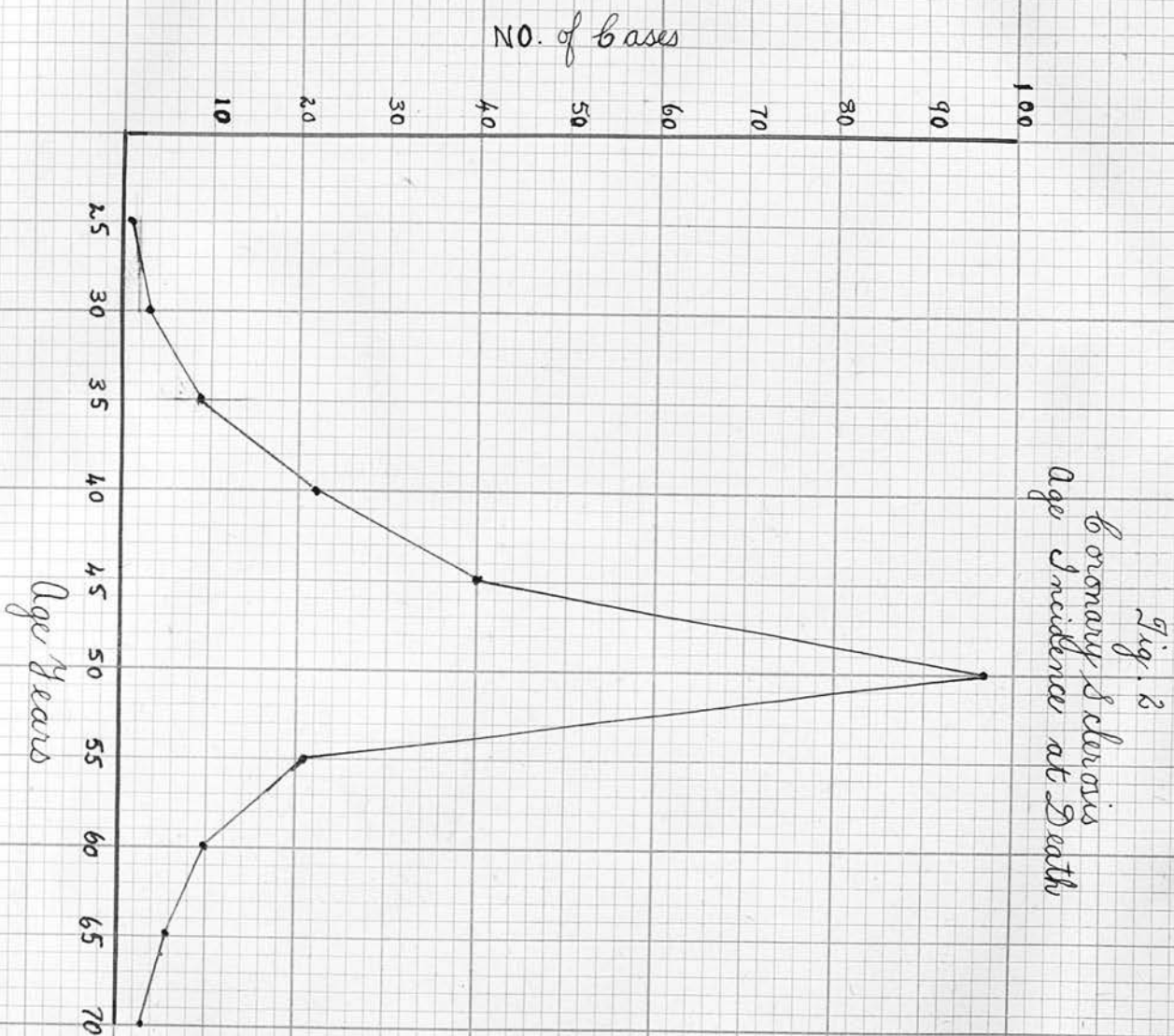


Fig. 3
 Coronary Sclerosis
 Age Incidence
 At Death in Males and Females

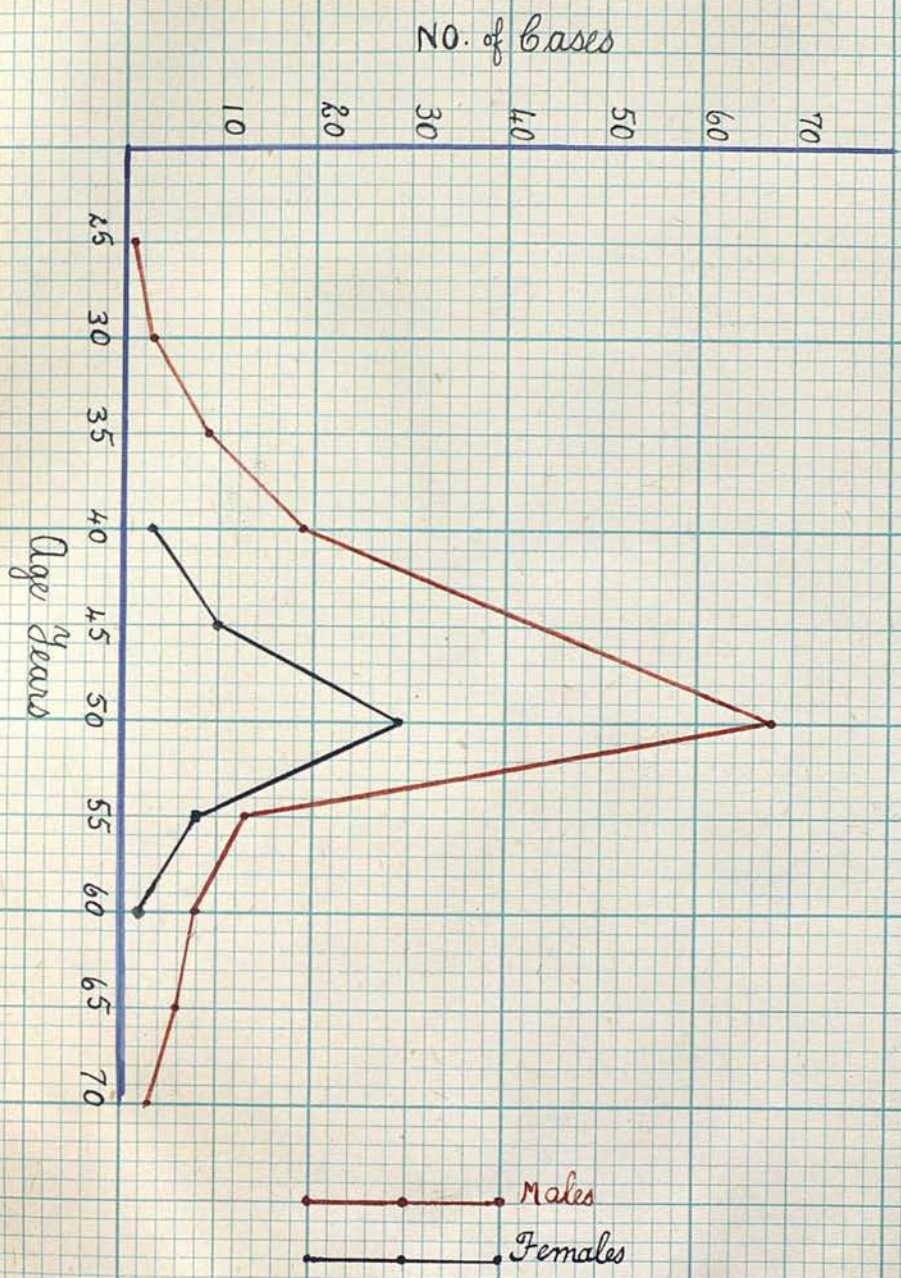


Fig. 4
Coronary Sclerosis
Age Incidence at Death
Males and Females

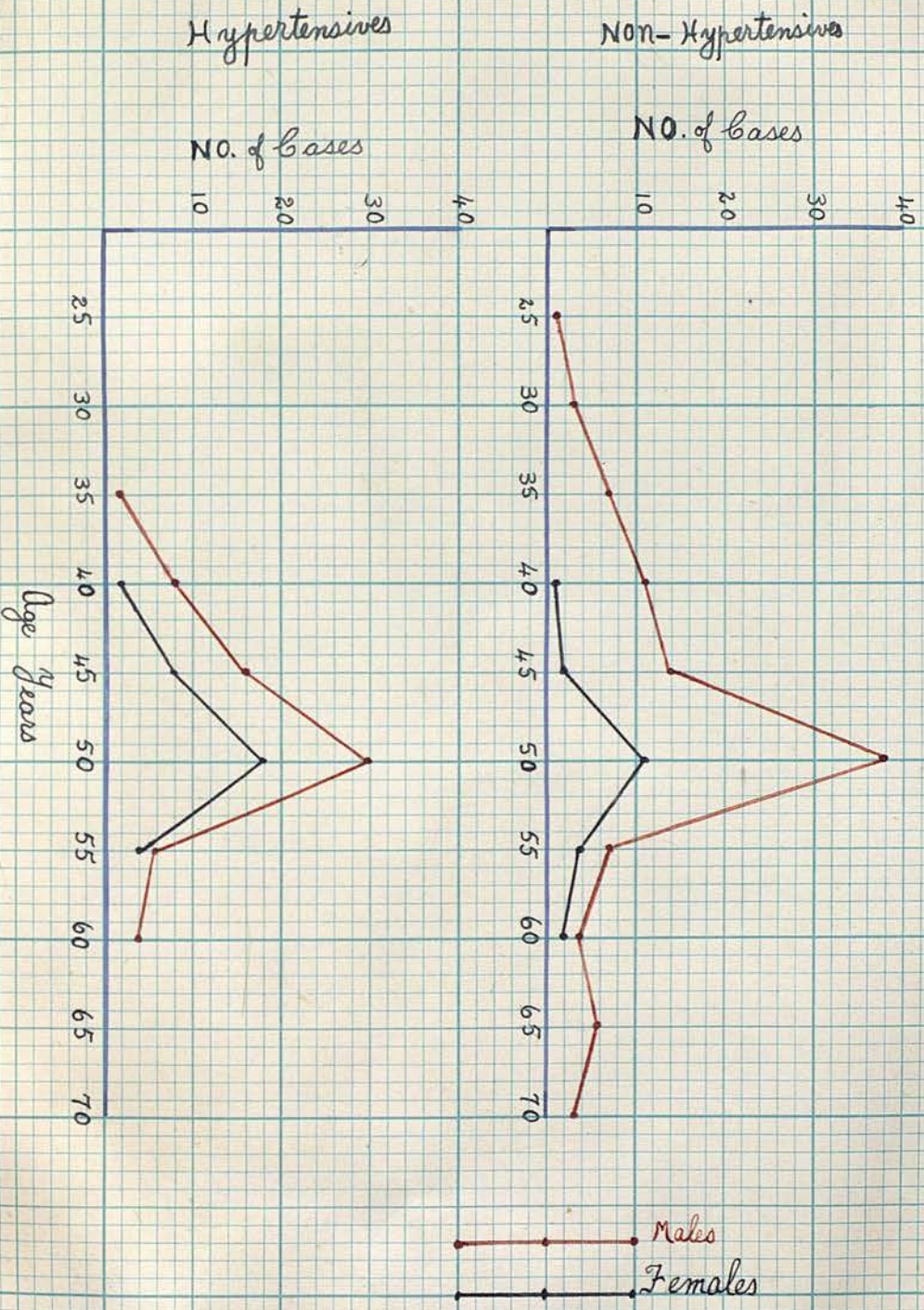
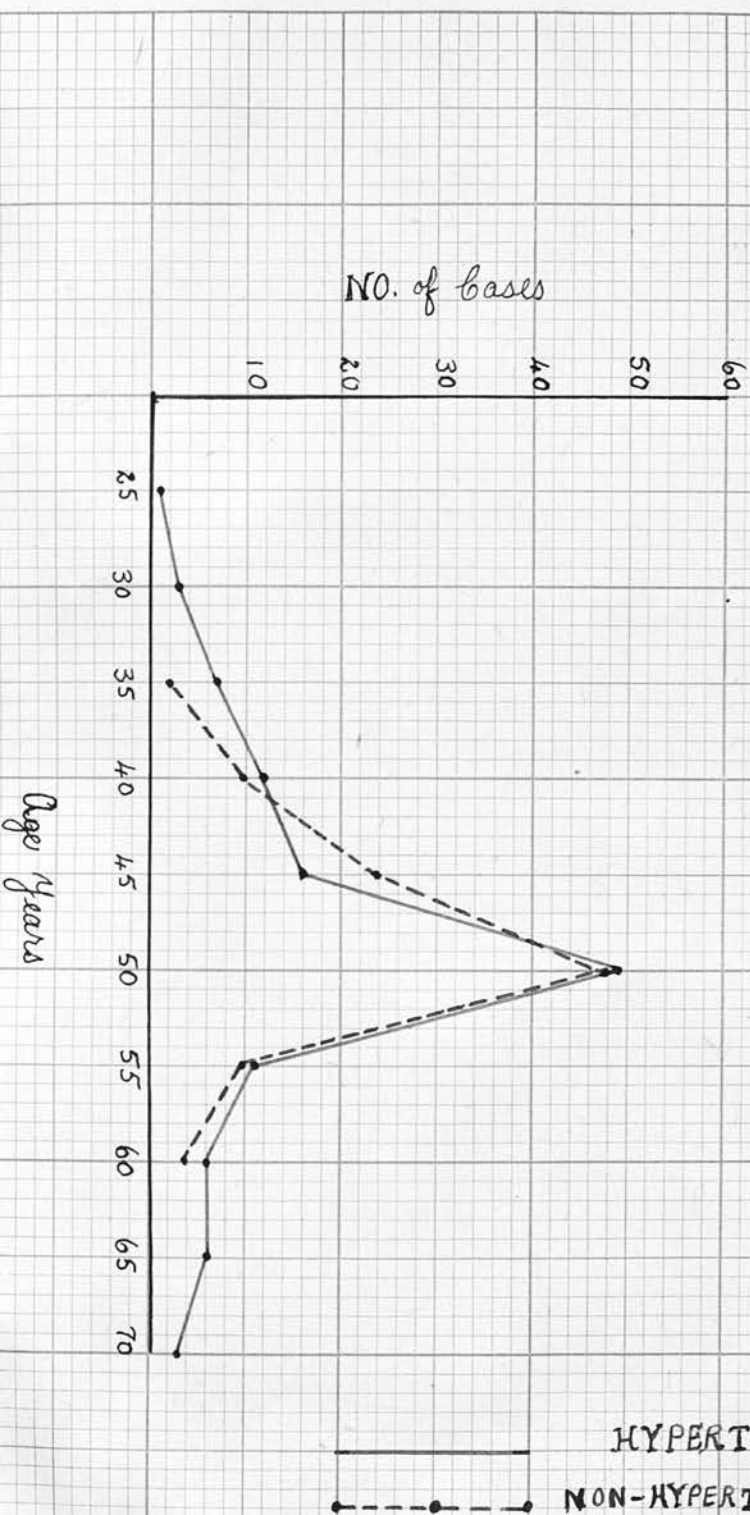


Fig. 5
Coronary Sclerosis
Age Incidence at Death
Hypertensives
and
Non-Hypertensives



- (c) The incidence of death at ages of 40 and 55 years was almost the same in either age group (5.7%).
- (d) The rise to the peak from the age of 45 years, as well as the fall from the peak to the age of 55 years was sudden, although more marked in the latter.

Fig.3. represents a contrast between age incidence of males and females at death.

- (a) In the male sex death occurred between the ages of 25 and 70 years.
- (b) In the female sex death occurred between the ages of 40 and 60 years.
- (c) In both sexes the maximum death rate occurred at the age of 50 years; 42.5% in males and 54% in females.

Fig.4. illustrates the age incidence at death in the hypertensive and non-hypertensive groups in both sexes.

- (a) In the hypertensive group the rise to the peak was sharper than in the non-hypertensive group.
- (b) In both groups the fall from the peak was sharp.

Fig.5. shows a contrast between the age incidence of hypertensive and non-hypertensive persons at death.

- (a) The outstanding finding was that, irrespective of the presence or absence of hypertension the fall from the peak was sudden in both groups.
- (b) In the hypertensive group the rise to the peak was slightly sharper than in the non-hypertensive group.
- (c) The span of life was longer in the non-hypertensive group.

From /

From the point of view of age incidence in general, a comparative study between this series and Clawson's series (1939) of 923 cases, shows that in the latter series the maximum death incidence occurred in the sixth and seventh decades of life. The combined maximum death rate in those two decades was 58.9%. In the present series the maximum death rate occurred about ten years earlier than in Clawson's series and the combined maximum death rate between the ages of 45 and 50 was higher than in Clawson's series; namely 64.7%

THE HYPERTENSIVE AND NON-HYPERTENSIVE GROUPS

Incidence

Sex

Race

A comparative study between the present series and Clawson's cases (1939), and Boloubeck's cases (1945) is self criticising as tabulated below:-

SERIES	GROUP	No. of Cases	Per Cent	Males	Females	M : F
Present	Hyper-tensive	98	46.2	66	32	2.1: 1
	Non-hyper-tensive	114	53.8	94	20	4.7: 1
Clawson (1939)	Hyper-tensive	414	45.0	326	88	3.7: 1
	Non-hyper-tensive	509	55.0	442	67	6.6: 1
Holoubeck (1945)	Hyper-tensive (Whites)	129	30.6	101	28	3.6: 1
	Hyper-tensive (Negroes)	294	69.5	185	109	1.7: 1

It should be noted here that in Holoubeck's cases the majority of autopsy records included negroes (63.6% of the total records).

However, the following facts are disclosed:-

- (1) Hypertension is a common finding with coronary sclerosis.
- (2) It is of a commoner occurrence among the black than the white race, although it is a known fact that the black race is less susceptible to coronary disease than the white race.
- (3) Its incidence among males to females is higher in the white than in the black race.
- (4) The incidence of hypertension among Egyptians is equal to that recorded by Clawson, one and a half times that recorded by Holoubeck among the whites, and about two thirds that recorded among the negroes.

In this connection it is noted that American Negroes have higher blood pressures than African Negroes, and that crossing with the whites seems to increase the incidence of hypertension in American Negroes (Weiss and Prosmark 1938) and Kesilman (1941).

2. Sex Incidence:

Male cases were 160 and female cases 52; an incidence of 3.1 : 1 respectively.

3. Race - Heredity.

Out of 212 cases there were 6 Greeks, 5 Italians, 5 Syrians, 3 Turks and 3 Sudaneze. The rest were Egyptians.

A family history of angina pectoris was obtained

in /

in 62.8% of the cases.

4. Body Constitution:

Among the whole series there were:-

65 Obese
23 Well nourished
93 Average Weight
31 Under weight.

Among the hypertensive group of 98 cases, there were 40 obese; (40.8%); 22 were females and 18 males.

5. Habits:

Not much was gained in this respect.

In over 60%, a history of smoking was obtained, but was not ascertained whether it was moderate or heavy. However, nicotine staining was marked on the fingers of 15 on both hands. None was noticed on the hands of the females.

A history of drinking was denied in almost all cases where history was obtained.

6. Social Conditions:

Among males there were 36 of the professional classes, 4 retired, 38 labourers, 18 carters, 10 cabmen, 8 doorkeepers, 6 waiters, 6 porters, 10 without certain work, 8 farmers, 10 unemployed and 6 persons of unknown occupation.

The females were all housewives with the exception of 4 maids and 3 washerwomen.

7. Associated Diseases.

Diabetes /

7. Associated Diseases:

Diabetes mellitus occurred in 30 cases, 21 males and 9 females, an incidence of 14.1%.

8. Season:

The cases were received over nine years in the following seasons as follows:-

Winter.....64
Autumn.....60
Spring.....47
Summer.....41

9. Interpretation of Lesions and Clinical History:

Three groups of patients were found.

- (a) Group died with symptoms, 132 cases - 62.3%
Past history was available in 64 cases only.
- (b) Group died without symptoms, 48 cases-22.6%
Past history was available in 27 cases only.
- (c) Group found dead, 32 cases, - 15.1%
Past history was available in 6 cases only.

A contrast study of the cases with known past histories is illustrated in the following table:-

FINDINGS	64 cases with symptoms at death	27 cases without symptoms at death	6 cases found dead
Degree of sclerosis	Sclerosis ++	Sclerosis +++	Sclerosis ++ +++
No. of anginal attacks	1 - 3	2 - 4	3 - 5
Duration	5 years	3 years	3 years
Predisposing factors	Effort 60.7% Ordinary activity 39.3%	Effort 37.5% Ordinary activity 62.5%	26 in bed 6 on road
Angina in family	34 cases	14 cases	6 cases

N.B. Fibrosis of the heart occurred in all the above cases as well as in 25 other cases; an incidence of 57.5%. The fibrosis was not related to the vicinity of the coronary arteries.

From a study of this table the following points may be observed:-

- (i) The more the degree of sclerosis was present the less prone were the patients to develop symptoms at death. This suggests that a fibrosed myocardium is less sensitive to ischaemia.
- (ii) Symptomless death was likely to occur with less effort.
- (iii) Expectancy of life was shorter in patients with a severe degree of sclerosis.

10. Distribution of Coronary Sclerosis in the Main Arteries.

This is shown in the following table:-

Artery	No. of Cases	Per Cent
Left Coronary	108	51.0
Right Coronary	77	36.3
Right and Left	27	12.7

In this connection it was noted that:-

- (a) A severe degree of involvement of the right coronary artery (+++) occurred at the age of 55 years in 15 cases (14.4%).
- (b) A lesser degree of sclerosis (++) was found in the hypertensive group (70 cases), i.e. in 71.4% of these cases.

11. Symptoms:

Symptoms were encountered in 62.3% of the cases.

Pain was the most common symptom. Its site was mainly in the precordial region. Other sites of pain were the back, epigastrium, neck, jaw, shoulders, arms and wrists. Two patients complained of cramp-like pains in the legs, and one patient complained of severe pain in the throat while he was taking ice cream. He eventually died in convulsions in less than 30 minutes, and a suspicion of food poisoning arose before the diagnosis was settled^e at autopsy.

12. Mode of Death

Death was almost instantaneous in 9 cases, three having dropped dead while walking in the street. In 15 cases death was stated to have occurred within fifteen to twenty minutes. These very sudden deaths occurred in both the hypertensive and non-hypertensive groups, 8 and 16 cases respectively.

D I S C U S S I O N

Heberden in 1768, first described a "disorder of the heart", "Marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it".

Parry, in 1799, wrote, "The rigidity of the coronary arteries may act proportionately to the extent of the ossification, as a mechanical impediment to the free motion of the heart; and though a quantity of blood may circulate through these arteries-----yet there may probably be less than what is requisite for ready and vigorous action". (East and Bain 1948).

Heberden's angina is due to a relative anoxaemia of the myocardium, as a result of sclerosis and narrowing of the coronary arteries. The kind of danger belonging to it is undoubtedly sudden death.

"Angina" as a technical term came down from classic times, and for almost two centuries before Heberden it was used in England to designate cases of Quinzy or sore throat in which a feeling of strangling and anxiety entered (Dock 1939).

As the immediate pathological basis of angina pectoris is atherosclerosis of the coronary arteries attended with severe narrowing of their lumina, it is justifiable at this stage to review some significant aspects of atherosclerosis.

I. CORONARY ATHEROSCLEROSIS

A. Definition of Atherosclerosis in General.

Marchand, in 1904, defined atherosclerosis as a diseased condition of the arterial wall with thickening of the inner layer, accumulation of lipoid substances, degenerative changes often with calcification, and distortion of the artery (Cowdry, 1933, p. 355).

"In atherosclerotic vessels of the aged, mucin is found in increased quantities, both in the intima and in the media. Hyaline is another substance observed principally in the intima of vessels that have undergone atherosclerotic changes. The thickening and hardening of the arterial wall may thus be due to infiltration of several totally different substances." (Cowdry, 1933, p. 7.)

B. Points of Importance in the Pathology of Coronary Atherosclerosis.

(i) The Nature and Site of the Lesion in Atherosclerosis.

The lesions are markedly found in the intimal

layer /

layer. These are described as thickened fibrotic areas invariably showing fatty changes, also collections of fat bearing macrophages, so-called atheromatous "abscesses", foci of calcification and occasionally definite bone formation (Horn and Finkelstein, 1940).

(ii) Distribution of intramural capillaries,
their origin and Formation of Atheromatous
Abscess.

It was noted that the intramural capillaries of the coronary system were most abundant at sites where fibrosis of the intima was not marked and also in the neighbourhood of fatty plaques (Leary 1934, 1935, 1936, and Horn and Finkelstein, 1940), but they diminish considerably or even become absent in the vicinity of ossified and calcified plaques, and also at sites of "abscesses". The formation of atheromatous abscesses is held due to a diminished blood supply and to the absence of fibrosis, (Leary 1934, 1935, 1936 and Horn and Finkelstein, 1940).

Leary is of the opinion that thrombosis follows the rupture of such abscesses. He also maintains that since the tendency to fibrosis of the original intimal lipoid is greater in younger individuals, the formation of these abscesses, therefore, does not occur. The apparent conclusion from this observation is that thrombosis of the coronary arteries is of a more frequent occurrence in older than in younger persons.

However /

However, such abscesses are stated by Horn and Finkelstein to have been encountered in younger persons. In this connection, they maintain that, irrespective of age group, the formation of an atheromatous abscess is inversely proportional to the degree of vascularisation, and that necrosis must ensue whenever intimal vascularisation does not keep pace with fatty change and fibrous intimal thickening. They emphasised this fact by finding the combination of an atheromatous abscess in the vessel wall at the site where vascularisation was absent, and the non-occurrence of such an abscess in the same vessel on the opposite side of the abscess where vascularisation was rich.

Jucker, 1935, on the other hand, holds that a toxic agent is responsible for such foci. However, since it was indicated by Bruening that in a normal artery the intima is nourished from the lumen, whereas in atherosclerosis, the intima draws its blood supply from the vasa vasorum, it is the view of observers like Horn and Finkelstein and Klemperer and Otani (1931) that the role of the blood supply is the more significant factor. Accordingly Ramsey (1936) and Winternitz et al (1938) advanced the view that vascularisation of the intima may precede the development of atherosclerosis, and that the lipoids deposited within the intima may be derived from extravasated blood through

the /

the new vascularisation. Horn and Finkelstein and Leary and other workers maintain that vascularisation of the intima only occurs in response to the degenerative process of atherosclerosis and never precedes it. Horn and Finkelstein, therefore, conclude that vascularisation, in a sense, is a nutritive and reparative reaction to atherosclerotic degenerative process.

The intramural vascularisation was demonstrated to originate mostly from the adventitia, and to a lesser extent from the lumen, (Gross et al 1934, Von Glahn 1936 and Horn and Finkelstein). A third origin was advanced by Klemperer and Otani, that intramural capillaries were derived from embryonic mesenchyme tissue retained by the arterial intima.

(iii) Role of Calcium in Experimental Atherosclerosis.

Han and Lewis (1934), produced experimentally intimal sclerosis of the coronary arteries of rats 28 days old. Fourteen rats were used in the experiment. Seven of them were given in addition to their regular diet activated ergosterol.

Autopsy was performed on 3 rats which survived for 22 days, and the one rat which died on the 21st day. The essential lesions found were:-

The media showed a heavily calcified ring and a marked proliferation of tissue inside the media. The newly formed tissue encroached on the lumen in varying degrees/

degrees, and the new lumen which had formed in each vessel was found lined with very cellular endothelium, which gave the impression of recent intimal proliferation. In many instances the lumina of the vessels were almost completely obliterated.

They maintain that the intimal lesions as well as the medial lesions are the result of nothing more than the inability of the blood to retain on certain occasions all its calcium in solution.

The cardiac muscle in these rats showed areas of strophy associated with increase of connective tissue and some haemorrhage.

These results were interpreted with a previous experiment by Ham in 1932, in which he gave the rats one single dose of activated ergosterol. The striking feature of this experiment was the extreme rapidity with which calcification of the media of the aorta and coronary vessels and cardiac muscle occurred. This calcification, according to Ham, is not related to degenerative changes in the recipient tissues. He concluded that the rapidity of formation together with massiveness of the calcification, suggested strongly that the prime factor was the inability of the serum to retain all its calcium in solution.

II INCIDENCE OF CORONARY ATHEROSCLEROSIS

It is a general agreement that atherosclerosis is
the /

the most frequent disease of the coronary arteries.

It is stated that Mönckeberg's (1915-1916) studies of coronaries in fatalities in the first World Great War showed that in 652 autopsies, coronary atherosclerosis was found in 535 cases (Cowdry 1933, p.437). This gives an incidence of 54%.

Holoubeck (1945), in a series of 1,045 cardiac deaths, found an incidence of 20.7%, but it was noted that the majority of her cases were negroes who formed 63.6% of the series; a large proportion which actually, on a racial factor, decreased the incidence of atherosclerosis. However, in her series, it was indicated that atherosclerosis was a commonly associated cardiac complication amounting to 33.6% in another group of death (hypertensive). The combination of these two groups raises the mean incidence of atherosclerosis to 27.2%; an incidence half as big as that given by Mönckeberg.

White (1943), however, has observed that coronary sclerosis varies in its relative incidence in different parts of the world, largely according to the frequency of such other causes as rheumatic heart disease, hypertension and syphilitis aortitis.

The incidence in the present series agrees with Mönckeberg's findings.

III. DISTRIBUTION OF ATHEROSCLEROSIS

(A) Incidence of Atherosclerosis of the

Right /

(A) Incidence of Atherosclerosis of the
Right and Left Coronary Arteries.

According to Mönckeberg (1915-16), in a series of 114 cases, the left coronary artery was affected earlier than the right one.

Bork (1926), opposed this statement and maintained that the left is no more often or earlier affected than the right coronary artery.

However, most investigators agree with Mönckeberg.

Horn and Finkelstein (1940) stated that severe atherosclerosis occurs in the major vessels with equal frequency.

In a series of 100 autopsies, they found that the anterior descending branch of the left coronary artery was involved by atherosclerosis in 85 instances, the left circumflex branch 71 times and the right coronary artery 76 times. However, they are of the opinion that these three major vessels are involved with equal frequency.

The present series is in accord with Mönckeberg's statement.

(B) Incidence of Atherosclerosis in the
Divisions of the Branches of the
Coronary Arteries.

With regard to the divisions of the branches of the coronary arteries, it was found in the Horn and Finkelstein series that the primary division of the anterior descending branch was involved in 48 cases, whereas in 26 instances this was present in the

branch /

branch of the left coronary artery which runs to the obtuse margin of the heart. It was also found in the same series that the divisions of the left coronary artery were involved 175 times and those of the right coronary artery 40 times.

Such incidence was not studied in the present series.

(C) Incidence of Atherosclerosis in the Intermuscular Branches.

Horn and Finkelstein (1940) observed atherosclerosis of the intermuscular branches of the coronary arteries in four out of one hundred cases.

This was also observed by Saphir et al (1935) and Glomset (1938), and other authors.

Sutton and Brandes (1930) found that this incidence was much greater than already stated, as they had encountered the condition in 42 of their 49 cases of coronary atherosclerosis.

Such incidence was not studied in the present series.

IV. AGE INCIDENCE

According to Rössle (1919), age incidence of atherosclerosis is expressed as follows:

Table overleaf

Age Incidence (Rössle 1919)

AGE	INCIDENCE PER CENT
15 - 20 years	10.6
20 - 25 "	10.8
25 - 30 "	22.7
30 - 35 "	27.0
35 - 40 "	34.1
40 - 45 "	31.6
45 - 50 "	50.0

Mönckeberg (1915 - 16) in a series of 114 cases recorded the youngest age as being 17 years old with affection of the left coronary artery, and 19 years old with affection of both coronary arteries.

Juvenile atherosclerosis, in a comparatively younger age, was recorded by other observers.

Khoo (1943) described a case of a Chinese aged 12 years, who on routine flourescopic examination of the chest revealed a markedly elongated and tortous aorta, and prominence of the two ventricles especially the left.

Guild et al (1938), described two other cases, one in a girl aged 9 years and 9 months, and the other was aged 9 years and 10 months.

According to Zacks (1943), review of literature disclosed five cases of medial coronary sclerosis in infants from birth to 27 months. Zacks also quoted

Benda /



Benda, who described atherosclerosis in a case of 12 years old.

Sprague and Orgain (1935), reported on a case aged 15 years old. Therefore, from a glance in the above table and series, there is no place of an element of surprise, when at times younger cases than usually met with are encountered.

Nevertheless, the condition usually manifests itself in a recognisable proportion from the fourth decade onwards; but it is not a disease of the aged, as one occasionally finds at autopsies arteries with minimal lesions. This observation is maintained unanimously.

Schlesinger (1940), gives an incidence of 40% in males over 55 years of age. This incidence is noticed to be less than encountered at a younger age period of only five years, as illustrated by Rössle.

Levy et al (1934) in a series of 762 cases of angina pectoris found that coronary atherosclerosis had increased at all ages, noticeably between the ages of 25 and 44 years and that the increase predominated in males.

In the present series maximal age incidence was between 40 and 55 years of age, with the peak at age of 50 years.

V. SEX INCIDENCE

All authors are in agreement that the male sex predominates.

An explanation is offered on the basis of an anatomical /

anatomical peculiarity, which is believed to establish the sex incidence. This anatomical peculiarity lies in the greater thickening of the intima of the coronary arteries in the male than in the female sex. It is present throughout life and demonstrated from birth.

The predilection of atherosclerosis of the coronary arteries is due to their possessing an intima varying in thickness from 100 to 600 per cent that of the media and averaging 26% in new born males and 8% in females (Dock 1946).

The following table gives the incidence in both sexes according to three authors:-

Author	Year	M	F
Clawson	1939	2.5	1
Gordon and Bland	1946	3.0	1
White	1946	3.0	1

However, Gordon and Bland stated that in older people of 70 years and over there was no significant sex difference in occurrence of coronary changes.

Sex incidence in the present series is in agreement with the average incidence given in the literature.

VI. RACE

Atherosclerosis is a disease most frequently encountered in the white race. This is generally agreed upon. It is also agreed that negroes are less susceptible to the disease (Moritz and Zamcheck, 1946).

The incidence of whites to negroes in Holoubeck's series, as calculated, was 1.5 : 1.

It is of interest that it was noted in Holoubeck's series that the incidence of hypertension in the negroes was more than double the incidence in the whites - 2.3 : 1 respectively.

The conclusion drawn from this observation is that although the black races show a higher proportion of arterial hypertension, yet they are less susceptible to coronary artery disease than the whites.

VII. HERIDITY

This factor is obtained in a recognisable proportion of people. Mortensen (1925) in an analysis of 300 cases of atherosclerosis, found a family history in 67.5%. O'Hare, quoted by Boyd (1943), obtained a family history in 68% of cases. One patient whose father died of apoplexy and mother of cardiovascular-renal disease, had nine brothers and sisters all of whom had died of apoplexy, and he himself had already had a stroke.

Zacks (1943) related a hereditary factor in a

case /

case of a 19 year old Jewish male who died of a coronary occlusion. His mother was hypertensive and a family history of myocardial disease was also obtained.

These examples are some of many encountered in literature and every-day practice.

VIII. BODY CONSTITUTION

Overweight and obesity are, according to many authors, factors which over tax the heart in health and disease.

French and Dock (1944), in a survey of 100 fatal cases of coronary artery disease, reported on overweight as a predisposing factor in 91% of the cases.

According to Moritz and Zamcheck (1946) the relation between body weight and sudden cardiac death is illustrated as follows:-

Body Weight	Sudden Cardiac Death per cent
Very light	2.9
Underweight	16.0
Normal	17.0
Overweight	41.0
Very heavy	23.0

IX. DIABETES AND CORONARY SCLEROSIS

Diabetes is by no means an associated disease found in atherosclerosis in general. However, it has been also encountered in association with coronary sclerosis in varying proportions.

Warren (1930), in a survey of 270 autopsies found reports of coronary sclerosis in 124 cases, an associated incidence of 45.9%. Coronary sclerosis was of a marked degree in 52 cases.

Nathan (1932), studying 100 diabetics at autopsy found an associated incidence of 41% of coronary sclerosis. He also remarked that 39 of the patients were more than 50 years of age.

The apparent mechanism in this connection seems to be a predisposition of diabetics towards atherosclerosis, an abnormal cholesterol metabolism being in question.

This association was observed in 14.1% of the cases in this series.

X. ADDITIONAL EXTERNAL FACTORS

Most authors agree that there are such factors which may precipitate death, in hearts with sclerosed vessels. These factors are recognised as effort, emotion, cold, alcohol, tobacco and even over-eating (Boas 1942, Smith 1943, Laplace 1946).

Master (1947), on the other hand is of the opinion that stress and strain of modern life, or the use of tobacco or alcohol do not supply a significant evidence in the production of disease of the coronary/

coronary arteries. That may be so in some cases. However, the evidence produced by Master himself in respect of the increase of the incidence of coronary disease in the past few years is undoubtedly not only attributed to a lengthened span of life, but also to the strain and stress of modern life. A faulty interpretation may arise from a false conception in a group of cases in which symptoms or even death are not related to effort, emotion or other such factors, as it is sometimes encountered.

In clearance of some doubt, the following statistics and reports are self-criticising:-

(A) Effort and Asymptomatic Death in Angina Pectoris.

There are encountered every now and then cases of sudden death in which symptoms were minimal or even absent (Laplace 1946, Poe 1947).

Poe, reporting on fatal coronary artery disease in young men from records of one army hospital, found 9 cases between the ages of 22 and 40 in which death occurred suddenly. 7 patients had no symptoms, 1 had mild dyspnoea, and 1 vague abdominal distress. All the 9 cases showed atherosclerosis of the two coronary arteries and their branches. In only two cases fresh thrombosis was found.

These observations are in agreement with the present series. It is also evident that the number
of /

of the arteries involved by the disease, and the degree of the sclerosis are of significance in determining the severity of pain in some cases, or even its absence on other occasions.

It may be of interest to record the conditions under which death in these 9 cases occurred. This is shown in the following table:-

Poe's (1947) Cases
Asymptomatic Death under Various Conditions.

No. of Cases	Conditions under which Death Occurred
3	Lying in bed.
1	Beginning morning exercise.
2	During moderate exercise.
2	Immediately after violent exercise.
1	After alcoholic drink.

These asymptomatic groups of cases may give no warning either to the patient or, sometimes, to the medical attendant. In that respect it is worth quoting Laplace (1946). "Disappearance of recurrent anginal pain may be considered a gratifying occurrence, but it should not be forgotten that the underlying coronary sclerosis is progressive, and that progression rather than regression of the disease may have caused the apparent improvement, and that it is not rare for sudden death to occur shortly after a

patient/

patient has been "cured" of his angina".

It is of special interest in this connection to record another, but larger, group of cases encountered in young soldiers during the second World War, in whom death, though it had ushered in with fatal seizures, had occurred under almost the same conditions as described by Poe. These cases were reported by Moritz and Zamcheck (1946), and are illustrated in the following table:-

Moritz and Zamcheck (1946)

Symptomatic Death Under Various Conditions

No. of Cases	Conditions under which Death Occurred
15	Sleep.
21	Inactivity - subject awake.
35	Ordinary activity.
29	Strenuous exertion.

The interpretation of these figures indicates that fatal seizures, in the majority of cases (71%), occur either during rest or ordinary activity. This is rather alarming, and classes sufferers from angina pectoris as potential subjects, under all circumstances, to death.

In the present series, effort was a significant factor in causation of symptomatic death.

(B) Emotion.

Emotional /

(B) Emotion

Emotional states such as fear, anger, and other forms of excitement are recognised causes precipitating sudden death in heart disease.

Many examples are found in the literature.

To recall two important instances: The death of John Hunter is one known to the medical profession.

King Philip V of Spain died suddenly when told that the Spaniards had been defeated. At autopsy a rupture of the heart was found.

Few examples of that type were encountered in the present series.

(C) Effect of External Heat and Cold on Patients with Angina Pectoris.

The effect of general and local application of heat and cold on exercise tolerance of 21 patients; (20 males and 1 female) was studied by Fredberg et al (1944). The exercise was stopped immediately a typical attack of angina was developed. Electrocardiographic evidence was also produced on the development of the attacks.

These studies have supported the concept that coronary artery vasomotor changes, probably reflex in origin, exert a contributory influence on precipitation of attacks of angina pectoris, and that heat acts as a coronary vasodilator and cold as a vasoconstrictor, or at least preventing vasodilatation. It was also noticed that patients who exercised in a warm room

while /

while holding an ice cube in one hand, developed pain. Such is probably the experience of many patients with angina pectoris who, although warmly clothed, only the face being exposed, have more attacks of pain in the winter than in summer. This remark is consistent with the seasonal mortality in the present series.

The suggestion made by these observers is that local application of heat enables many patients with angina pectoris to do considerably more work in a cold atmosphere. It is also prophylactic against anginal attacks.

XI. DURATION OF LIFE IN ANGINA PECTORIS

According to White and Bland (1932), the duration of life after the onset of angina pectoris is tabulated as follows:-

White and Bland's Cases

Duration of Life after Onset of Angina Pectoris	No. of Cases
4.4 years	213 (dead)
5.1 years	273 (living)
Average Duration of Life after Onset of Angina Pectoris	
4.9 years	Total 486

They /

They also mentioned 3 patients who had angina pectoris for over 20 years; 2 of these were dead and 1 was still alive over 21 years after the onset of his trouble.

In the present series the average duration of life after the onset of anginal attacks was 3 years and 8 months, the longest being 5 years. (History was available in 45.3% of the cases).

XII. CAUSE OF DEATH

The anatomical findings in these cases are consistent with the theory that angina pectoris is the result of paroxysmal, relative, myocardial ischaemia. The reduction of the coronary blood supply is undoubtedly mainly caused by atherosclerosis of the coronary arteries obstructing their lumina.

A severe degree of coronary atherosclerosis may result in an irrecoverable state of myocardial ischaemia and death. The ischaemic theory is held by many observers, among whom are Keefer and Resnick (1928), and Blumgart et al (1940).

In support of the ischaemic theory Reisman et al (1940) produced electrocardiographically by anoxaemia changes similar in all respects, except for the heart rate, to those observed in the same patients suffering from angina pectoris during attacks induced by exercise.

The myocardial fibrosis observed in the present series may be considered as evidence of the ischaemic theory /

theory in a recognisable proportion of cases.

Apart from coronary atherosclerosis as the main factor predisposing to myocardial ischaemia, Harrison (1935) and Wearn (1938) have pointed out that cardiac hypertrophy interferes with the oxygen diffusion into the cardiac muscle and its nutrition. Such a factor was encountered in the present series, (the hypertensive group).

The theory that coronary spasm may result in reduction or complete obstruction of the coronary flow and subsequent myocardial ischaemia is hardly acceptable. Sclerotic rigid vessels do not possess the quality of spasm (Blumgart et al 1940). It is possible then that the inability of such vessels to dilate during increased cardiac activity may be a significant factor contributing to the inadequate blood supply.

-----oOo-----

ACUTE CORONARY OCCLUSION

Case Material:

This group of coronary artery disease formed 85 cases; 60 occurring in males and 25 in females; an incidence of 23.1%.

The cases were received over a period of nine years according to the following three tables as regards the number of deaths per annum, seasonal incidence and place of death.

No. of Deaths per Annum

Year	1939	1940	1941	1942	1943	1944	1945	1946	1947
M.	6	8	2	5	3	8	10	7	11
F.	2	3	1	2	2	1	4	4	6

Seasonal Incidence

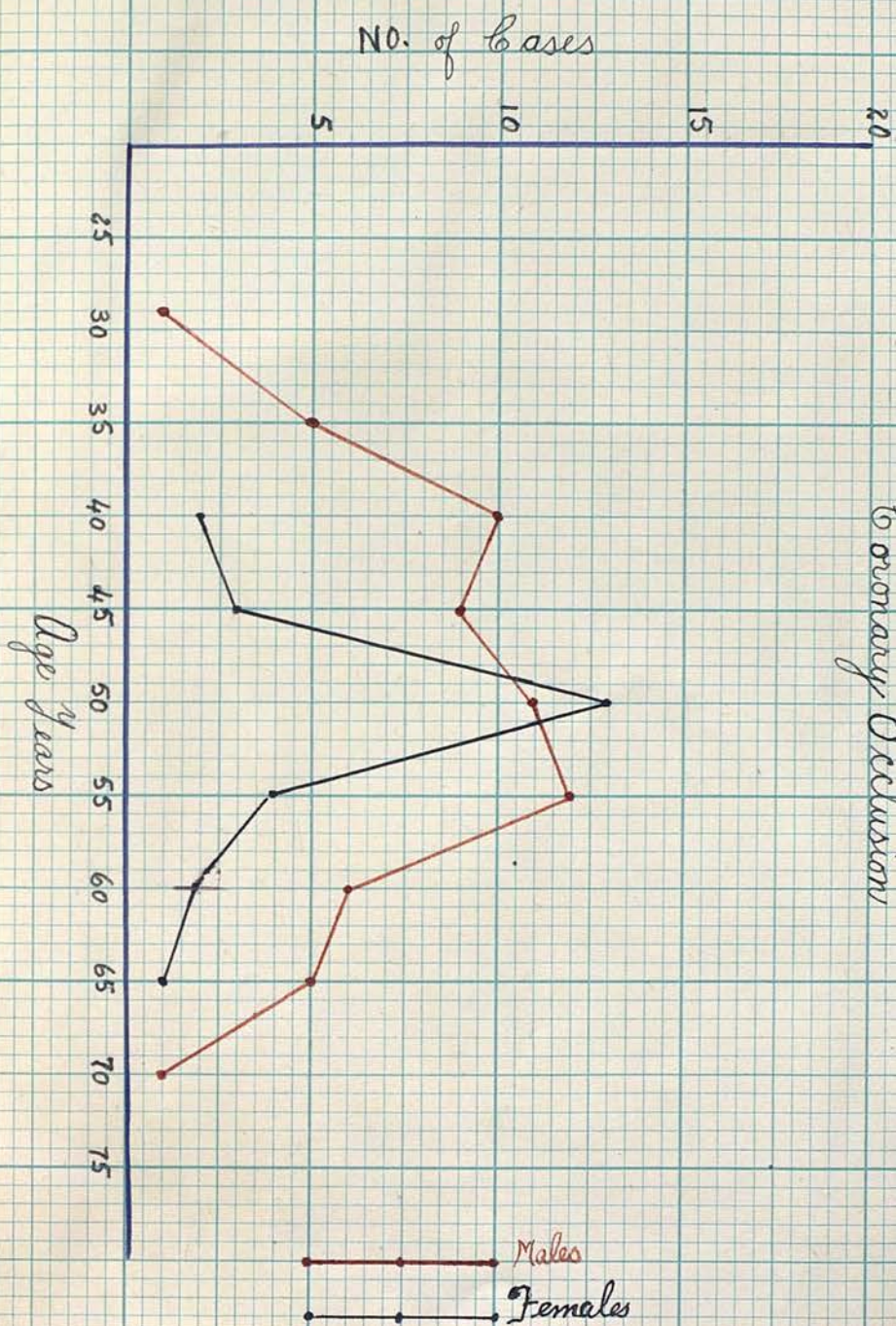
Season	Autumn	Winter	Spring	Summer
No. of Cases	22	36	15	12
Per Cent	25.8	42.4	17.6	14.2

Place of Death

No. of Cases	Sex	Hospital	Died in Public Places	Died at Home	Dead on the Road
60	M	4	9	31	16
25	F	3	1	18	3
85	Per	8.2	11.7	57.6	22.5

Fig. 6

Deaths
in
Coronary Occlusion



1. Age Incidence:

In this series the age incidence ranged between 29 and 70 years of age, the average age for males being 50.6 years and for females 50.8 years. The youngest male was 29 years and the oldest 70 years. The youngest female was 40 years and the oldest 65 years.

It was noticed that the deaths in males began 11 years earlier than in females and extended 5 years longer. The major incidence of death in males occurred at the age of 55 years, and both the rise to the peak and the fall from the peak were slow, in a stair-like fashion.

In the female sex, the major incidence of death occurred at the age of 50 years and both the rise to the peak and the fall from the peak were sharp and sudden. The peak in the females was slightly higher than in the males and occurred five years earlier. Deaths at ages of 40 years and under occurred in 18 cases; an incidence of 21.2%. Only one death occurred in females and males at ages of 65 and 70 years respectively.

A graphic representation is illustrated in fig: 6.

2. Sex Incidence.

There were 60 males and 25 females; an incidence of 2.4 : 1.

3. Race. /

3. Race:

All cases of males were Egyptians with the exception of 2 Greeks, 1 Italian and 1 Armenian.

The female cases included 1 Syrian and the rest were Egyptians.

4. Body Constitution:

Among the male cases, 16 were obese, 7 underweight and the rest of average build.

Among the female cases, 9 were obese, 2 underweight and the rest of average build.

Therefore, the males who were obese formed 26.7% and the females who were obese formed 36% of the cases.

5. Habits.

Smokers:- Males: 5 were heavy smokers (8.3%)
10 moderate smokers, (16.6%)
45 did not smoke (75.1%)

Females: 3 moderate smokers (12%)
22 non-smokers (88%)

Alcoholics:- Among the males 11 were moderate drinkers (18.3%)
The rest were teetotalers.

Alcohol was denied among the females.

6. Social Conditions:

Among the males there were 14 of the professional classes, 2 retired, 9 cabmen, 9 labourers, 6 farmers, 2 doorkeepers, 11 unemployed, and 8 persons of unknown occupation.

The females were all housewives.

7. Family History:

There was a definite family history of coronary artery disease in 19 cases: 13 males and 6 females; an incidence of 22.4%.

In this connection, an example is given. A lawyer, 45 years old, travelled 12 hours to see his dying father. The son died on the same day with coronary occlusion. The father was later known to have survived his attack.

8. Associated Diseases:

(A) Hypertension. On a pathological basis, estimation of cardiac hypertrophy, as previously stated in connection with coronary sclerosis, was considered indicative of hypertension.

There were 17 males and 8 females classed as hypertensive; an incidence of about 30.2%.

(b) Diabetes Mellitus. Among men there were 7 diabetics. Among the women there were 3 definite diabetics and one doubtful; an incidence of 11.8%.

9. Sites of Occlusion:

Evidence of recent occlusions were found as follows:-

Left coronary artery.....50 cases

Right coronary artery.....27 cases

Left and right coronary
arteries.....8 cases.

The incidence of occlusion of the left to the

right /

right coronary artery was 1.8 : 1.

It was also noted that the anterior descending branch of the left coronary artery was the one most frequently involved. However, it was found severely occluded by old thrombosis in 7 cases, and yet an apical anterior infarction was encountered due to a recent thrombosis of the right coronary artery. Such incidence suggests a collateral circulation having been developed at an earlier date.

10. Nature of Occlusions:

- (i) Thrombosis was encountered in 27 cases and was easily recognised by the naked eye.
- (ii) Intramural haemorrhages were encountered in 58 cases.

11. Symptoms:

These were in the following order.
Severe precordial pain, dyspnoea, epigastric pain, nausea, vomiting and collapse.

12. Mode of Death:

Without exception death was rapid in all cases, the shortest duration was about 30 minutes and the longest 5 to 6 hours.

Of the hospital cases, 5 died on reception; 3 of these were transferred from villages over a distance of 15 kilometres and were diagnosed as food poisoning.

The /

The remaining 2 cases died immediately after a preliminary examination, no clinical signs being recorded. The last 2 cases were transferred 3 kilometres in town, three and four hours after fatal symptoms.

Undoubtedly transference of patients in these cases, especially on the rough village roads, has added to the risks of the disease.

13. Conditions under which Death took Place.

In 53 patients the fatal attacks occurred during work, 26 died in bed and 6 were found dead on the road.

14. Mortality Rate in First Attacks and Duration of Life after First and Second Attacks.

These are illustrated in the following table:-

Mortality Rate	No. of	Per
First attacks	Cases	cent
	15	17.6
Duration of Life (years)	No. of	Per
after First Attacks	Cases	cent
6 months	28	32.9
1 year	12	14.2
2 years	9	10.6
Duration of Life (years)	No. of	Per
after Second Attacks	Cases	cent
3 years	6	7.1

In the remaining 15 cases either a history was wanting all together or none was available.

The average duration of life after the onset of

coronary /

coronary occlusion was 1.4 years.

15. Pulmonary Embolism as a Cause of Sudden
Death in Coronary Occlusion.

This was recognised as the cause of death in seven cases; an incidence of 8.2%. Emboli were found to have originated from intraventricular thrombi.

In this connection it was noted that death in four cases occurred within three to six hours from the onset of symptoms.

In the other three cases the history was not available.

C O M P A R A T I V E S T U D Y

1. Incidence:

It is noted by several authors that the disease is generally on the increase. Master (1947) has marked an increase in the prevalence of coronary artery disease and, more particularly, acute coronary occlusion. This is attributed by him to a lengthened span of life notably in the past 50 years due to reduction in infectious diseases of childhood and adult life, advances in medical knowledge, and improvement in public health and sanitation.

Accordingly more fatalities from this disease

would /

would be expected in the coming years. He quoted a hypothetised expectation of life - that by 1960 a boy might expect to live 75 years and a girl to 80 years (Piersol, 1940). Expectation of life at the present, according to Dublin and Latka (1937) is 70 years at birth. This was based on a study of the mortality rate among the industrial policy holders of the Metropolitan Life Insurance Company, New York, over 25 years from 1911 to 1935.

Moritz and Zamcheck (1942), in an analysis of postmortem records found atherosclerosis and thrombotic occlusion in 25% of the cases, and incomplete occlusions in 55% of the cases.

French and Dock (1944), in a survey of 100 fatal cases of coronary artery disease, found that arteriosclerosis was the basis in 80 cases. Recent myocardial infarction was demonstrated in 15 out of the 80 cases; an incidence of 18.8%.

Gordon et al, (1946) in an analysis of their series found that coronary occlusion was more prevalent among private than among hospital patients.

The incidence of coronary occlusion in the series under discussion is in accord with the general view.

2. Age Incidence:

Coronary occlusion was originally regarded as a disease of late life, occurring commonly about the

ages /

ages of 45 to 60 years, but since the clinical diagnosis of the disease has been made with increasing frequency and accuracy, recent reports have shown that it is not uncommon among younger persons.

Reitman et al (1942) surveying the literature on coronary occlusion under the age of 40 years, found a total of 221 cases. Of these, 34 were below 30 years of age and only 4 below 20 years of age; an incidence of 17.2%.

In this series death at ages of 40 years formed 21.2%.

Newman (1946), recorded a series of 50 cases in soldiers between the ages of 20 and 35, of whom 40 died.

Zacks (1943), reviewing the literature, revealed 8 cases of coronary thrombosis between the ages of 10 and 20 years. In 6 cases, diagnosis was confirmed at autopsy. He also described a case of coronary thrombosis occurring in a 19 year old Jewish boy which, at autopsy, revealed a fresh thrombosis occluding the left circumflex branch immediately beyond its point of origin.

Still, the above records should be regarded as exceptions to the rule.

Levine and Brown (1929), in a series of 145 patients recorded an average age of 57.8 years. The highest /

highest incidence in their series was between 60 and 69 years, though between 50 to 59 years of age it was just as high. Only three patients were under 40, and one patient above 80. The average in females was slightly under the total average, being 57.4 years.

Conner and Holt (1930), in a series of 287 cases encountered one third of the cases under 51 and three quarters under 61 years of age. They, therefore, concluded that coronary thrombosis must be regarded as essentially a disease of early middle life rather than of later life.

Enklewitz (1934) stated that coronary thrombosis occurred predominantly in the 6th and 7th decades.

The conclusions drawn from the above observations are that the disease is of a high incidence in early middle life, as well as in the fifth and sixth decades, although a number of cases occur in young and still fewer in elderly individuals, and that those cases occurring in the younger age group are probably on the increase.

With regard to coronary thrombosis occurring in the aged, Willius (1937) reported on a case of a man aged 92 and produced electrocardiographic evidence in support. In this connection, he stated that he had seen coronary thrombosis in over 90 years of age, but only once.

In the present series the oldest patient was a

male /

male of 70 years of age.

3. Sex Incidence:

The male sex predominates, for the same reasons already discussed in connection with coronary sclerosis.

The following table gives the incidence in both sexes, according to eight authors.

AUTHOR	YEAR	M.	F.
Parkinson and Bedford (clinical cases)	1928	13.3	1
Parkinson and Bedford (autopsy cases)	1928	7.5	1
Levine and Brown	1929	3.2	1
Conner and Holt	1930	5.7	1
Gordon et al	1939	3.0	1
Smith et al	1942	6.6	1
Fisher and Zukerman	1946	2.7	1
White	1946	3.0	1

The average incidence of males to females according to these records is 5.6 : 1. However, according to four of the above authors, the average incidence is 3 males to 1 female, which is the ratio usually given in the literature.

In the present series this ratio was 2.4 : 1.

4. Body Constitution:

It /

4. Body Constitution:

It has been claimed by some authors that obesity is a factor in coronary occlusion (Levine, Connor and Holt).

The following illustrates this relation according to two authors:-

AUTHOR	YEAR	OBESE per cent.
Smith et al	1942	35
Moritz and Zamcheck	1946	23

These findings are in accord with the present series, although the females in this series formed the larger proportion.

However, it was noted by many observers that the immediate prognosis and consequent complications in coronary occlusion are less favourable in obese patients.

The dreaded immediate risk is acute heart failure due to adiposity of the heart.

Hypostatic pneumonia heads the complications in these cases and is attended with a high mortality rate.

5. Habits and Occupation:

According to White (1946), tobacco, alcohol, tea and coffee are probably without direct influence in predisposing to coronary disease.

Smith et al stated that alcohol was insignificant

in producing coronary occlusion.

Emerson (1929) referring to Wynne and Guilfooy, found an incidence of 18.9% of coronary artery disease among cigar makers and tobacco workers. According to these observers, 20.7% of coronary disease was found among garment workers, a proportion still higher than is found among the previous groups. Railroad track and yard workers formed 17.6% and blacksmiths 15.8%. Painters and paper hangers, machinists and clerks and book-keepers formed 14.9%, 14.1% and 14% respectively. The smallest incidence of coronary disease was found among teamsters and drivers (10.3%) and saloon-keepers and bar-tenders (10.1%).

It would appear, therefore, from the above statistics, in respect of the varied nature of these occupations, that the evidence in support of an habitual or occupational factor is not very significant. However, few authors advanced some theories in this respect, claiming that nicotine particles in the form of dust or fumes predispose to thrombo-angiitis obliterans, coronary sclerosis and hypertension (Wright and Moffat, 1934 and English et al 1940).

In the present series alcohol does not seem to be a significant aetiological factor. Only 18.3% were moderate drinkers among the males and none

among /

among the females.

Among the males there were 8.3% heavy smokers, 10% moderate smokers and the rest did not smoke. Female smokers formed 12%. However, they were all moderate smokers.

It is interesting to note that smoking was almost just as prevalent among females as among males.

6. Heredity.

Heredity, as a predisposing factor of coronary artery disease has been known for generations.

An account of the views of some authors to this effect has already been referred to in connection with coronary atherosclerosis. To add to the previous authors, Levine and Brown found that many patients gave a family history of coronary disease.

The present series is only an example of many.

7. Seasonal Incidence.

In accordance with the seasonal incidence observed in this series, Hoxie (1940) found that in the subtropical climate of Los Angeles, acute attacks of coronary occlusion were definitely more frequent in winter than in late summer and early fall. The same observation was also noted on attacks occurring in early spring.

However, he adds that in comparison with a series reported from a north temperate region it seems likely that there are factors other than the

degree/

degree of seasonal temperature variation. Frequency of infections during the colder months is considered by him the most potent of these factors.

8. Sites of Occlusions.

It has been found by most authors that occlusions of the left coronary were commoner than those occurring in the right vessel. This is illustrated in the following table according to two authors:-

Author	Left Coronary	Right Coronary
Fisher and Zukerman (1946)	1.93	1
Moritz and Zamcheck (1946)	2.50	1

It was also found that the anterior descending branch of the left coronary artery was more frequently involved than the left circumflex branch as shown in the following table; and thus has been named "the artery of coronary occlusion".

Ratio of Occlusion of Anterior
Descending to Circumflex
Branch of the Left Coronary
Artery.

Author	Ant.Descend.	Left Circumflex
Horn & Finkelstein (1940)	1.2	1
Schlesinger (1940)	1.4	1
Parkinson & Bedford (1928)	2.4	1

The present series is in accord with these
findings/

findings.

On the other hand, Horn and Finkelstein (1940), opposed the first statement as they recorded, in their series, occlusions of the right to the left coronary artery at the ratio of 1.1 : 1 respectively.

9. Mechanism of Coronary Artery Occlusion.

Generally speaking, intramural haemorrhage and thrombosis are the two more frequent findings encountered in this mechanism.

Arteriosclerotic narrowing of the coronary arteries is the pathological basis of all such occlusions (Hamman 1926, Wolff and White 1926, Boyd 1928 and Horn and Finkelstein 1940). Clawson (1939) stated that thrombosis of a nonsclerotic vessel was rare.

In rare cases, an embolus from a vegetation on a valve may block a coronary artery or one of its branches. When an embolus blocks a main vessel, death occurs with dramatic suddenness. Such occlusions were not encountered in the present series.

Syphilitic aortitis of the aorta sealing the mouths of the coronary arteries is another recognised cause of coronary occlusion. This is discussed later in this chapter.

Occlusions by intramural haemorrhage or thrombosis are the subject of this discussion.

A. Intramural Haemorrhage. The general accepted view by many authors is that intramural haemorrhage/

haemorrhage in a sclerotic vessel is of a more frequent occurrence than thrombosis. Horn and Finkelstein (1940) reported a ratio of 1.7 : 1 respectively. Emphasising this fact, they stated that even when a thrombus was found unassociated with intimal haemorrhage, they had been able to demonstrate its origin at a point in the arterial segment of the artery which disclosed the most advanced sclerotic changes and narrowing.

Paterson (1938) disclosed 32 instances of intramural haemorrhage in 37 occlusions.

Keeping this fact in mind, it is therefore unjustifiable clinically to speak of the term "thrombosis" in this connection. A more correct clinical diagnosis would be the use of the term "acute coronary occlusion" as it appears that the ultimate differentiation must rest entirely upon the histological interpretation (Horn and Finkelstein 1940).

The formation of an atheromatous abscess has already been described. The degenerative process in an atheromatous abscess impinges upon the sub-endothelial tissue, and as the newly formed capillaries in relation to the "abscess" are fragile, rupture of these capillaries frequently occurs, and the artery may thus become narrowed or even occluded by the resulting haematoma.

Intramural haematomata of few days' duration may undergo changes, such as red cell degeneration and fibrinoid transformation (Horn and Finkelstein).

B. Thrombosis. Thrombosis should be considered as an incident in coronary occlusion, as in many cases it occurs subsequent to intramural haemorrhage. However, when the degenerative process in an atheromatous abscess is extensive, it was found by Horn and Finkelstein, that very little haemorrhage was sufficient to produce rupture of the intimal layer and to initiate thrombus formation. They also maintain that intramural haemorrhage is always coexistent with degenerative changes in the intima. These changes in the form of fatty degeneration, loose separation of the intimal fibres, or scattering of erythrocytes impinge upon the endothelial surface. Such changes, whether originally induced, or intensified by the haemorrhage ensue endothelial damage and thrombus formation.

10. The Role of the Collateral Circulation in Coronary Sclerosis and Occlusion.

This may be reviewed under the following headings:-

A. Evidence of the Presence of a Collateral Circulation.

This was demonstrated by:-

(1) Blumgart et al (1940), using a lead-agar material injected in the coronary arteries in autopsy material.

(2) Prinzmetal et al (1947), using red cells and glass spheres of known dimensions in perfusion studies on human hearts studied postmortem.

B. Observations/

B. Observations on the Rate of the Development and Extent of the Collateral Circulation.

(1) Age of the Patients.

Blumgart et al considered that the rate of the development and extent of the collateral circulation were not related to age, as inter-coronary anastomoses were not present in the hearts of even senile patients when sclerosis was present.

On the other hand, other observers, Gross (1921), and Campbell (1929), stated that the collateral circulation developed with the advance of age.

(2) Conditions under which Collateral Circulation Developed.

Blumgart et al maintained that the collateral circulation developed only when and where it was needed. The anastomoses were found to have increased in extent in the presence of coronary sclerosis.

In addition to the above findings, it was also found that there were other factors which needed the establishment of a collateral circulation. Arterial hypertension, valvular obstruction or insufficiency, thyrotoxicosis, infection, exercise, a rapid ventricular rate, and even emotion were all factors which increased the work of the heart and its metabolic demands; thus calling for the development of an adequate collateral circulation. If such demand was not gratified, the "coronary reserve", so to speak, was reduced by those factors.

C. Mechanism/

C. Mechanism of Development of Collateral Circulation.

This mechanism at the present time seems a subject of debate. According to Blumgart et al, the pressure in the arterial segment distal to the narrowed or occluded zone becomes greatly reduced. This would favour an increase in the size of the channels which communicate with other normal or relatively uninvolved arteries containing blood under higher pressure, and thus a functioning collateral circulation develops.

Winternitz et al (1938) maintain that the Vasa vasorum may take part in formation of a collateral circulation.

D. Functions of the Collateral Circulation.

According to Blumgart et al and other investigators, the functions are of two folds.

(a) Furnishing a sufficient blood supply to the myocardium distal to the site of narrowing or occlusion, thus preventing to some extent myocardial damage.

(b) Allowing the cardiac muscle to meet an extra demand of function whenever it is called upon to do so.

Accordingly it was found that in the presence of an adequate collateral circulation many patients with coronary sclerosis do not suffer from anginal pains, and even some patients with minimal coronary occlusions were without symptoms. This also applied to other conditions demanding increased action of the heart./

11. Mechanism of Production of Pain in Coronary Occlusion.

"Progressive reduction in the lumen of the coronary artery either before the formation of the thrombus or with a gradually growing thrombus" is maintained by Feil (1937) to be the most acceptable explanation.

Herrick (1935) holds that pain is possibly due to rupture of an atheromatous "abscess" with subsequent formation of a dissecting aneurysm in the wall of the involved coronary artery.

In the present series intramural haemorrhages were of a frequent occurrence. The pain, therefore, is likely to be caused by myocardial ischaemia due to sudden diminution of the calibre of the artery (Blumenthal and Reisinger 1940). These last observers also noted that since the obstruction is acute, but only partial, the pain will last only during the period of circulatory readjustment. The effect on the myocardium may be reversible if the obstruction is not complete, otherwise infarction may occur.

Katz et al (1935) and Martin and Gorham (1938) found that mechanical stimulation of the adventitia and periarterial nerves in experimental animals causes pain, even when the coronary flow is unobstructed. Blumenthal and Reisinger conclude that tension on the adventitia and its nerves, caused by the intramural/

intramural haemorrhage, may thus be the excitor factor of pain in man.

12. Associated Diseases.

A. Hypertension. Hypertension has been reported by many authors as a common finding in coronary thrombosis (Cowdry, Conner & Holt, White and Bland). The low blood pressure observed after the development of coronary thrombosis is explained on an inadequacy of the injured myocardium and reduced ventricular output (Cowdry).

As to the significance of hypertension as a major aetiological factor in the production of coronary thrombosis it has been noticed that although hypertension is common among the female sex and negro race (East & Bain 1948), yet coronary thrombosis is not of a frequent occurrence in women and negroes as would be expected.

Perhaps what is more important in this connection is the observation made by White and Bland (1932) that the ultimate prognosis in coronary occlusion has not been affected materially by hypertension. It has been noticed only slightly more frequently in those who died within one year after their seizures than in those who survived four years.

B. Diabetes Mellitus. The intimate relationship between diabetes, abnormal cholesterol metabolism, and the early onset of coronary sclerosis and thrombosis is brought up by many observers.

Reitman et al (1942) reported on a 20 year old diabetic/

diabetic patient who had an attack of coronary occlusion. In this case they produced an electrocardiographic diagnosis in support.

Enklewitz (1934) in an analysis of 92 cases of diabetic patients found 26 cases of coronary thrombosis among them; an incidence of 28.3%. He maintained that in both diabetic and non-diabetic patients, coronary thrombosis occurred predominantly in the 6th and 7th decades. In another analysis of 100 diabetic patients over the age of 40 he found that 34 showed coronary thrombosis and 45 coronary sclerosis. Although he admitted that both thrombosis and diabetes over the age of 40 are manifestations of a degenerative vascular disease, yet he stated that coronary thrombosis occurred twice as frequently in diabetic as in non-diabetic patients. He also referred to Warren (1930) and Root (1928), who had described in diabetics lesions of atherosclerosis of the pancreatic vessels. Barron (1925) found advanced atherosclerosis of the pancreatic arteries in cases of coronary sclerosis.

In describing the distribution of atherosclerotic lesions, Fishberg (1931) listed the kidney, spleen, pancreas, liver and brain in the order of the frequency of these lesions.

Levine and Brown (1929) and Conner and Holt (1930) reported an incidence of 23% and 10% respectively.

In/

In the present series diabetes mellitus occurred in 11.8% of the cases.

13. Complications.

A. Infarction of Organs. According to Garvin (1942) about 60% of cases of myocardial infarction had one or more infarcts in the lungs, brain, kidneys, extremities and intestines.

The incidence of infarction of organs due to other diseases of the heart is given in the following table:-

Incidence of Infarction in
Heart Disease.

<u>Diseases.</u>	<u>Infarction per cent.</u>
Subacute bacterial endocarditis	66.6
Coronary artery disease:	
with myocardial infarction	60.0
without do. do.	50.0
Rheumatic heart disease	50.0
Hypertensive heart disease	40.0
Syphilitic heart disease	40.0

B. Pericarditis and Coronary Occlusion.

According to Cowdry (1933), pericarditis is a frequent finding in coronary occlusion.

Levine and Brown (1929) noted pericarditis in 13.8% of their cases.

In the present series, pericarditis was not encountered as a complication. This may be due to the fact that death occurred before such a complication/

complication had time to develop.

14. Causes of Death in Coronary Occlusion.

A. Ventricular Fibrillation. In recent years the mechanism of death in coronary occlusion has been the subject of an intensive study by some workers.

Ventricular fibrillation has been advanced as the primary cause of death in many of these cases.

Smith (1939) reported on a case of a male, aged 40 years, with a history of 9 hours coronary artery occlusion. The patient died after a convulsive attack. An electrocardiogram, taken a few minutes after convulsions, showed ventricular fibrillation. At autopsy a recent thrombus was localised in the anterior descending branch of the left coronary artery.

Thompson (1941) proved the same electrocardiographically in a male aged 66 years, who eventually died during the process of recording his electrocardiogram. This patient had, 23 weeks previously, developed an attack of coronary occlusion. At post mortem the left coronary artery was found occluded in several places. No evidence of fresh thrombosis was found. However, before his death the patient developed an attack of palpitation and finally went into ventricular fibrillation.

Thompson stated that his case was the seventh on record.

B. Pulmonary Embolism from Intraventricular Thrombosis.

B. Pulmonary Embolism from Intraventricular Thrombosis. This is a recognised cause

of death in coronary thrombosis. It is sometimes the cause of sudden death in this disease, as it was found in the present series. It accounted for not less than 3.2%.

In contrast with this, Eppinger and Kennedy (1938), in 200 autopsied cases of coronary thrombosis, encountered sudden death due to pulmonary embolism from intraventricular thrombosis in 6.5% of the cases.

15. Sudden Death and Coronary Occlusion.

Coronary occlusion has been recognised by a majority of authors as a common cause of sudden cardiac death.

On the other hand, Boissard and Duguid (1938) were of the opinion that it was not. Reporting on their records of the Cardiff Royal Infirmary, during seven and a half years, they stated that the condition was seldom if ever fatal, and that even in the most severe cases death was usually delayed for some hours or even days after the development of the lesion. They also stated that even when one of the large coronary branches was involved, resulting in widespread infarction of the left ventricle, the victim usually survived for many hours after the onset of the symptoms. They described a case in which more than half of the wall of the left ventricle was necrotic, and yet the patient lived for at least 12 hours. They maintain that unless occlusion/

occlusion is widespread so as to arrest practically the whole circulation of the heart at once, sudden death is hardly to be expected.

This statement may be true in some cases; but on the other hand, Eppinger and Kennedy in the same year, in a study of 200 autopsied cases of coronary thrombosis found:-

32% died suddenly
35.5% died of congestive heart failure
14.5% died of other causes.

However, Gross in 1921 stated that the anastomosis between the peripheral branches of the coronary arteries is quite adequate in old age, and that occlusion of a small branch is hardly likely to cause a significant deleterious effect.

That coronary artery disease in general is responsible for an appreciable proportion of deaths is expressed by Master (1947), as forming 8.5% of all deaths. He did not, however, state whether these deaths were sudden or not. In connection with cardiac fatalities alone, Master stated that coronary artery disease accounted for 30 to 50%.

Parkinson and Bedford (1928), in an analysis of necropsies in the London Hospital on 83 cases of cardiac infarction, including 7 cases of mortal coronary occlusion without infarction, recorded an incidence of 29% of cases brought to the hospital dead.

16. Mortality Rate and Coronary Occlusion.

A/

A mortality rate of 16.5% is given by Master (1947).

Levine (1945) quoted figures of 15-25%

Poe (1947) gave a mortality rate of 7.4% of coronary artery disease in general among all deaths from natural causes.

Master gave a mortality rate of 8% in first attacks.

Conner and Holt (1930) found a mortality rate of 16% in first attacks.

In the present series, the mortality rate in first attacks was 17.6%.

17. Duration of Life after the First Attack of Coronary Occlusion.

This is illustrated according to Conner & Holt in the following table:-

<u>Conner and Holt (1930).</u>	
<u>Duration of Life</u> <u>(years)</u>	<u>Living Patients</u> <u>Per cent</u>
1	75.0
2	56.0
5	21.0
10	3.4

They also mentioned one instance of a patient who lived 18 years after his first attack. This is, probably, the longest duration of life mentioned in the literature.

White and Bland (1932) on the other hand, recorded/

recorded a shorter duration of life after the first attacks or coronary thrombosis, as tabulated below:-

White and Bland (1932).

Duration of life after Onset of Coronary Thrombosis	No. Cases	Living Patients per cent
1.5 years	101 (dead)	
3.2 years	94 (Living)	
Average Duration of Life After Onset of Coronary Thrombosis		
2.4 years	195	

In the present series, the average duration of life after the first attack was 1.4 years. There were only 6 patients who survived a second attack over a period of 3 years; an incidence of 7.1%.

CORONARY OSTIAL OCCLUSION

Coronary ostial occlusion was the cause of sudden death in three cases.

C A S E M A T E R I A L

Case I

S.A.S., a male, aged 43, quarryman, single, was admitted to hospital on 3.3.40. with a severe attack of gripping pain in the lower part of the chest, radiating down the left arm to the level of the elbow, of one and a half hours' duration. He was working on the road when he experienced this pain coming on almost suddenly, though preceded by a sense of oppression in the chest for a few minutes. He vomited twice with the onset of the attack.

Previous History: The patient had five or six attacks of a similar nature, though considerably milder, during the last three months. Three of these attacks occurred during working hours. Each attack lasted for 15 to 30 minutes. No medical attendance was received during any of the previous attacks. Syphilitic infection was admitted 18 years previously.

Physical Examination: The patient was obese and markedly dyspnoeic. The temperature was 37.5; pulse rate 110 per minute; respiratory rate 28 per minute and blood pressure 120/80. The apex beat was in the 5th space, nearly 2cm. outside the mid-clavicular line. A soft systolic murmur was heard

in the mitral area, and the second pulmonic sound was loud. Fine crepitations were heard over the lungs posteriorly. The lower edge of the liver was felt one finger-breadth below the costal margin. The spleen was not palpable. Examination of the abdomen and central nervous system revealed no abnormality. No ankle oedema was present. Urinary examination showed no abnormality. Blood urea was 34mgm.%. The blood Wassermann reaction was positive (+++).

The patient, however, did not respond to any form of therapy, became increasingly dyspnoeic, and died $3\frac{1}{2}$ hours after admission to hospital.

Autopsy revealed the following: The heart was dilated and flabby. It weighed 410 gm. The left ventricular wall was 2 cm. in thickness and the right ventricular wall 0.7 cm. thick. There was a slight evidence of pericarditis revealed by the presence of a semi-granular exudate.

The aorta presented three puckered, almost rounded, patches of a pearly colour occupying the proximal 5 cm. of the intima. The proximal patch was of a fibrous consistency, 1.5cm. across, raised $\frac{1}{4}$ cm. above the intimal surface, mostly situated behind the left posterior aortic cusp, involving the cusp and completely occluding the mouth of the left coronary artery. The course of this artery distal
to /

to its mouth was patent throughout, though few atheromatous plaques were found. The other two patches were slightly larger in size and of a harder consistency, and one of them almost cartilagenous. Besides these lesions, the aorta showed atherosclerotic plaques in a moderate degree, but was elastic except in these three areas. The right coronary artery hardly contained any atheromatous lesion and was patent throughout.

The aortic cusps were slightly incompetent.

Histologically these patches showed fibroplastic poliferation involving the vasa vasorum, collection of lymphocytes and few plasma cells and necrosis in the media.

The myocardium, otherwise, presented no syphilitic involvement.

The lungs were acutely congested and oedematous. The liver was enlarged and congested, and the spleen was congested.

Case II

N.M., a male, Greek, aged 40, barman, single, was admitted to hospital on 9.12.43. with a severe vice-like pain over the left of the sternum, radiating up to the left shoulder and down the left arm, of one hour's duration. He was on service when he experienced the attack of pain and had to lie down, but without relief. A bout of severe coughing followed the pain and made it worse. On the way to hospital he vomited once and was noticed to have been very short of breath and turned pale.

Physical /

Physical examination: The patient was well nourished and gasping for breath. The temperature was 38; pulse rate 120 per minute; respiratory rate 35 per minute and the blood pressure could not be taken.

The heart was stated to be enlarged and faint.

The patient soon died after examination - less than two hours from his fatal symptoms.

Previous history: The patient was stated in the last three weeks preceding his death to have been weak and short of breath. He was off his duty twice because he took dizzy attacks and was shaky in the legs, but apart from this he carried out his work satisfactorily since his employment four years previously.

No history of a syphilitic affection was obtained.

Necropsy revealed the following: The heart was firm and weighed 360 gm. The wall of the left ventricle was 1.5cm. in thickness and that of the right ventricle was 0.5 cm. thick.

The aorta was thickly atheromatous. The orifice of the right coronary artery was completely occluded by an elevated scarred patch 2 cm. across and $\frac{1}{2}$ cm. thick, involving also the adjacent two cusps. The distal course of the right coronary artery was almost free of atherosclerotic plaques and quite patent. The left coronary artery presented a moderate degree of atherosclerosis /

atherosclerosis.

The aortic cusps were short, thickened and incompetent. Histologically syphilitic aortitis was evident.

The lungs were oedematous and congested. The liver and spleen were congested. The stomach was empty and mucous membrane presented a general hyperaemia and few petechial haemorrhages.

Case III

Z. El. S., a female, Sudaneze, aged 35, single, was admitted to hospital in collapse. The symptoms preceding collapse were severe epigastric and sternal pain and numbness in the left arm of nearly $3\frac{1}{2}$ hours' duration. She was beating a carpet in her house when she first complained of this pain but ignored it as it was not alarming then. However, the pain never left her but she continued her daily work and while going up the stairs to the roof surface with her laundry, she was heard screaming and was found lying on the stairs. She pointed to her chest, being unable to speak, expressing a severe pain there. Her mother, thinking that she might have injured her ribs, called for an ambulance and was taken to a First Aid Hospital where she was referred to a General Hospital. On the way to the hospital she was able to give an account of her first symptoms already described.

Previous history: No previous history of such an attack was available, but the patient was noticed to have been breathless and easily tired in the last six months.

Physical examination: The patient was well-covered and comatose. Breathing was laboured;

temperature /

temperature was 38; pulse rate 130 per minute and the blood pressure could not be taken. No form of injury was discovered externally, though nose bleeding was noticed. A lumbar puncture revealed no abnormality. The apex beat was not palpable. A faint blowing systolic murmur was heard in the mitral area and a doubtful diastolic murmur was suspected in the aortic area. Fine crepitations were heard over the bases of the lungs posteriorly. No other abnormality was detected in the other systems of the body.

The patient's condition grew worse and she died within the first hour of hospitalisation.

Necropsy revealed a dilated flabby heart. Its weight was 350gm. The right coronary orifice was occluded by a syphilitic plaque, similar to that of case II. Its size was 3cm. across and was also found involving the adjacent cusps. The aorta and the left coronary artery presented atheromatous lesions, but the course of the left coronary artery was patent. The aortic cusps were similar to those of the previous case. Another syphilitic patch was found in the aorta, 3cm. distal from the first, measuring 2cm. across.

The lungs were oedematous and congested. The liver and spleen were congested. The skull and its contents were found intact.

None of the cases above presented a syphilitic arteritis beyond the orifices of the coronary arteries.

D I S C U S S I O N

1. Incidence of Syphilitic Ostial Occlusion:

The condition was found three times in the whole series of autopsies of 3,250 cases, an incidence of about 0.1%.

Its incidence among syphilitic affections of the aorta and valves was 9.4%.

It was responsible for 0.9% of sudden cardiac death.

2. Age Incidence:

The condition was encountered in the three cases described at ages of 43, 40, and 35 years, an average age incidence being 39.3 years. Its occurrence at a comparatively early period before middle life suggests an early involvement of the syphilitic lesion. Death at the ages noted also suggests a rapid fatal progression of the disease. The condition occurred in the female sex at a younger age than in males, and it was noted in this particular case that it occurred in a black person.

3. Sex Incidence:

3. Sex Incidence.

Two cases occurred in males and one in a female, a ratio of 2 : 1 respectively. This suggests a heavier infection of syphilis in the male than in the female sex. Such a syphilitic sex prevalence was also noted in connection with aneurysm of the aorta. A discussion of this prevalence is referred to in the section on aneurysm of the aorta.

4. Race.

The condition occurred in an Egyptian, a Greek and a Sudanese.

5. Social Conditions.

All cases occurred in the working class, which may suggest either an ignorance of the condition, or improper previous treatment, if any had been secured.

6. Symptoms, Mode of Onset and Duration.

Severe, sudden, gripping and vice-like sternal pain radiating down the left arm occurred in the three cases; the numbness of the left arm in case III being probably an ameliorated type of pain. Pain referred to the left shoulder occurred in one case. Vomiting started with the onset of pain in one case, and after one hour from the onset of pain in one case. In this latter case the pain was also followed by a bout of severe cough. Nose bleeding occurred in one case and collapse in another. Dyspnoea, rapid pulse and /

and a noticeable rise in temperature occurred in all cases. The duration of symptoms was 2 to 5 hours before death had set in.

The symptoms were related to physical effort in two cases at least, anginal like, only of a much longer duration.

7. Past History.

In one, case I, six similar attacks of a mild type occurred within the last three months preceding fatal illness. In the other two cases, a history of breathlessness occurred within the last three to six months before death.

A syphilitic infection was admitted in one case eighteen years previous to death.

8. Body Constitution, Associated Diseases and Pathological Findings.

One patient was obese and the other two well-nourished. This suggests that overweight may be a burden on the heart and may have at least accounted for the breathlessness of some duration in two cases. In all cases there were no other associated diseases found at post mortem.

The determining cause of fatal issue in all cases was a syphilitic ostial occlusion. In no case did syphilitic arteritis involve the course of the coronary artery.

The /

The right coronary ostium was involved twice and the left one only once; a ratio of 2 : 1 respectively.

In all cases the heart exhibited a moderate hypertrophy, the weights being 410 gm., 350 gm., and 360 gm.

Dilatation of the heart was noticed in two cases.

The aortic cusps were incompetent and involved by the syphilitic process in all cases at their commissures.

No syphilitic myocarditis was found in any case.

Myocardial infarction did not occur with ostial occlusion of either the right or the left coronary artery.

Apart from the heart, all cases presented oedema and congestion of the lungs, also congestion of the liver and spleen. The liver was enlarged in one case.

C O M P A R A T I V E S T U D Y

Impaired circulation of the coronary arteries has received attention since Obrastzow and Straschezko in 1910 (Hammann 1922), and Herrick in 1912.

The combination of aortic insufficiency with coronary closure was commented upon by many authors as of frequent occurrence. Benson states that syphilitic occlusion accounts for most closures of

the /

the coronaries and that the effect on the heart is similar to other gradual closures of these vessels where sufficient time has elapsed for anastomosis with the other coronary to develop (Cannon 1930).

1. Observations on the Pathology of Syphilitic Coronary Disease.

Leary (1940) stated that in syphilitic lesions of the aorta, the vasa vasorum, under stimulation of the triponema pallidum, grow and branch and a fibroblastic tissue develops. The intima becomes greatly thickened in plaques. The growth in the connective tissue in the region about the aortic ring invades the orifices of the coronary arteries and those portions of the vessel which lie in the aortic wall. This narrowing may lead to essential occlusion of this portion of the vessel. However, the invasion of the coronary ostia is slow, and a collateral circulation to the heart, by anastomoses with pericardial vessels, bronchial arteries and the aortic vasa vasorum, carries on the coronary blood supply as Wearn (1928) and others have shown. When the coronary orifices are not particularly narrowed, early sudden death does not occur.

In opposition to the above mode of intimal invasion, McMeans (1931) is of the opinion that in syphilitic aortitis, the intima is involved primarily by direct infection from the blood stream, though he
also /

also admitted its invasion from the adventitia through the vasa vasorum.

Martland (1931) observed stenosis or atresia of the coronary ostia in about 30% of cases of syphilitic aortitis which resulted in death. Death from stenosis or atresia of the coronary arteries formed about 15%. He stated that Glahn (1925) attracted this attention by showing that often one or both coronary arteries may have their origin above the sinus of valsalva and thus become especially liable, by virtue of an abnormally high position, to scarring of their ostia. Martland also stressed the fact that coronary syphilitic arteritis is unusual to be found in the larger branches of the coronary arteries and is practically limited to a narrowing, or atresia, of their orifices by the lesion in the aortic wall.

On the other hand, Maher (1931), according to Bruenn (1934), has described changes of the larger branches of the coronary arteries similar to those found in the aorta. Moritz (1931), also reported cases with like findings.

In disagreement with the opinion of involvement of the distal branches of the coronary arteries by syphilis, Bruenn (1934), though accepting such a possibility, stated that this type of lesion is of questionable importance. He also quoted Warthin

(1922) /

(1922) who, though maintaining that localised syphilis of the smallest arterioles is an essential part of the general pathological condition of chronic or latent syphilis, added that, "Syphilis of the coronary arteries, in our experience, has been found much less frequently than anticipated from its frequent mention in the literature --- the larger branches of the coronaries only rarely show lesions that can be recognised as syphilitic".

Warthin again, (1931), emphasised the rarity of syphilitic involvement of the main divisions of the coronary arteries and stated that the smallest intermuscular branches of the coronaries are the most frequently involved.

The three cases under discussion presented no syphilitic involvement of any part of the course of the coronary artery, which is in agreement with the general opinion expressed above and by others such as Clawson and Bell (1927) and Leary and Wearn (1929).

The general agreement in respect of the incidence of the disease in the left and right coronary arteries is that the latter is the most frequently involved. This observation made by various authors is expressed in the following table, along with the present series which is also in accordance with the general findings.

Incidence /

Incidence of Syphilis in Right and Left
Coronary Arteries

AUTHOR	Year	R. Cor.	L. Cor.	R. & L. Cor.	No. of Cases
Clawson and Bell	1928	4	0	0	4
Reid	1931	3	1	1	5
Cannon	1930	1	1	0	2
Leary and Wearn	1929	2		1	2
Breunn	1934	9	4	26	35
Present series	1949	2	1	0	3

Breunn stated that, "There is no apparent explanation at the present time for the greater frequency and severity of right coronary orifice involvement."

2. Incidence.

Breunn gives an incidence of 4.6% of syphilitic involvement of the heart and aorta.

In Carr's (1931) cases, the incidence of coronary orifice involvement was 8.4%.

Martland (1931) gave an incidence of 14.9%.

Clawson and Bell (1927) reported an incidence of 19.8%.

Breunn series (1935) showed an incidence of 31.4%.

Sahir and Scott (1931) showed an incidence of 35.4%.

The mean incidence in the above records is 21.9%.

The /

The present series is in accordance with the above observations; an incidence of 9.4% of syphilitic ostial occlusion was encountered.

The incidence of involvement of the aortic valves by the leuitic process, according to Breunn, is 87.1%

In the present series, the aortic valve was involved in all cases.

3. The Incidence of Race, Age and Sex, and the Duration of Life in 62 Cases of Syphilitic Aortic Insufficiency

The following figures are according to Breunn (1934).

Syphilitic Aortic Insufficiency

	Normal Coronary Orifices (28 cases)	Stenosed Coronary Orifices (34 cases)
<u>Race:</u>		
Negro	7	16
White	20	17
Yellow	1	1
<u>Sex:</u>		
Male	23	27
Female	5	7
<u>Age:</u>		
(initial lesion)	25.3yr. (18-38)	23.6yr. (14-32)
(cardiac symptoms)	51.2yr. (32-69)	46.3yr. (25-62)
Time from first Cardiac Symptom to admission.	8.75 months (1 wk.- 3yr.)	8.2 months (1wk.- 3yr.)
Time from first Cardiac Symptom to death.	11.4 months (1wk.-3yr.)	9.9 months (2wk.-3yr.)
Duration of Life from primary lesion.	23.4yr. (4-35)	21.1yr. (5-39)

In addition most authors emphasise the relatively early age at which syphilitic coronary artery stenosis is found.

Burch and Winsor (1942) placed the average age at 40 years. The youngest case they found was in a person 20 years of age.

Clawson (1941) reported only one case occurring in the third decade.

Gorden et al (1942) reported the youngest case as being 32 years old.

Weinberg and Bessinger (1946) reported a case occurring in a female aged 28 years with coronary artery ostial stenosis and acute myocardial infarction.

From an analysis of the above series it will be found that the cases under discussion are in accordance with others' observations.

4. Symptoms.

These are also tabulated, according to Breunn (1934) in the previous cases, as follows:-

Chief Symptoms in Syphilitic Aortic Insufficiency

SYMPTOMS	Normal Coronary Orifices (28 cases)	Stenosed Coronary Orifices (34 cases)
Dyspnoea.....	18	20
Orthopnea.....	2	4
Paroxysmal nocturnal dyspnoea	3	0
Pain (Cardiac).....	10	13
Oedema.....	5	7
Cough.....	4	3
Weakness.....	2	6
Insomnia.....	1	0
No cardiac symptoms.....	2	4

Dyspnoea, cardiac pain and weakness form the chief symptoms in the above series of stenosed coronary orifices. These were also encountered in the present series, and in addition vomiting occurred in two cases and nose bleeding in one case. An interesting feature in the present series was some rise of temperature. Such was also found in a case reported by Breunn (1934) and Cannon (1930). In all these cases, there was no evidence of a myocardial infarction to account for the rise of temperature. A rise of temperature was also mentioned by Reid (1931) as being reported in literature in syphilitic affections generally, though in none of his cases was such recorded.

5. Mode of Death.

It appears from histories of cases reported in literature that death in these cases is fairly rapid in one group and sudden in another.

Bruenn (1934) reported a case of a 21 year old coloured girl who died 9 hours after symptoms, and another of a 35 year old widowed negress who died 12 hours after admission to hospital. However, he states that there is a slightly greater tendency for death to occur suddenly in these cases, but gave no actual figures as to the duration of time between fatal symptoms and death.

The mode of death in the previous 62 cases,
studied /

studied by him is given according to the following table:-

Mode of Death in
Syphilitic Aortic Insufficiency

Mode of Death	Normal Coronary Orifices (28cases)	Stenosed Coronary Orifices (34cases)
Gradual failure	6	5
Rapid failure	10	12
Sudden death	12	17

In the cases under discussion death was more rapid than in the two cases reported by Bruenn.

CHAPTER II

SPONTANEOUS CARDIAC RUPTURE

SPONTANEOUS CARDIAC RUPTURE

Spontaneous cardiac rupture occurred in 9 cases accounting for 2.6% of sudden cardiac death.

These cases were classified as follows:-

- (i) Rupture due to acute myocardial infarction.
- (ii) Rupture of cardiac aneurysm due to old standing coronary occlusions.
- (iii) Rupture due to adiposity of the cardiac muscle.

RUPTURE DUE TO ACUTE MYOCARDIAL INFARCTION.

Historical Review:

Reviewing the history of cardiac rupture by Benson et al (1933), it was stated that the earliest case was recorded by William Harvey in 1647. His patient was a nobleman who suffered with paroxysms of oppressive pains in the chest. Finally, "the disease growing more violent, he became cachectic and dropsical and at length, being violently oppressed in one of the paroxysms he died. In this patient, by reason of the circulation of blood being obstructed from the left ventricle into the artery, the very substance of the left ventricle itself, which seemed to be pretty thick and strong, had been ruptured and perforated with

a large opening where it had discharged the blood; for the foramen was sufficiently large to admit a man's finger".

According to Morgagni, Morand in 1732 had, "written and collected more than any other person ever did yet on the subject of rupture of the heart."

In 1765, Morgagni collected ten cases and added an observation of his own, "made upon a most powerful monarch". He did not know then that his own death would add another case to the history of cardiac rupture.

Blaud in 1820, according to Cruveilhier, collected four cases of rupture, and attributed them to a soft gelatinous degeneration of the myocardium.

In Dublin, Cheyne in 1818, Robert Adams in 1827, Townsend in 1832, Smith in 1836, Stokes in 1846, and Bellingham and Kennedy in 1849 attributed cardiac rupture to fatty degeneration. Adams described a rupture of the left ventricle in a female over 60 years old who, "died suddenly in a fit of vomiting".

In England, Williams in 1840 and Quain in 1850, though following the same lead, suspected coronary narrowing as might be the underlying cause.

However, as early as 1815, Hodgson, on reporting a rupture of the left ventricle where, "the degeneration of the structure of the ventricle had in one part proceeded to such an extent that the remaining fibres

were /

were unable to resist the force of circulation," had described an occlusion of the left coronary artery by calcareous deposit, though like all others overlooked thrombosis of the coronary arteries.

Barth in 1871, connected senile softening of the heart with atheroma of the coronary arteries, but this relation had already been definitely affirmed ten years earlier by Malmsten in 1861, who described "the clot in the coronary artery was undoubtedly old. The arterial obstruction had resulted in a slow softening of the muscular tissue. Finally rupture appears to have occurred slowly."

To mention others in this connection, Menard in 1878, and Winsor in 1880 are credited with illustrating this relation. Menard's valid description of coronary occlusion reads, "At one point, about 3cm. from the origin of the artery, one of these patches had so far protruded into the lumen of the vessel as to cause a coagulation of blood at that point, which coagulation had become adherent to the wall, thereby preventing the flow of blood through the vessel. The portion of the heart in which the rupture had occurred and in which the fibres were found degenerated corresponded to the territory supplied by the branches of this artery."

The condition from that time onwards was portrayed by many workers, to mention among whom were

Weigert /

Weigert (1880), Ziegler, Cohnheim and Huber.

C A S E M A T E R I A L

Case I.

M.M., a male, aged 60 years, a carter, suddenly collapsed while driving his cart along the road. He was immediately taken to the nearest chemist, but was found dead when a doctor examined him fifteen minutes later.

Previous to his fatal issue he had complained in the last two years of not less than three or four attacks of pains over the heart or in the stomach region. The pains were moderately severe on the last two occasions, namely nine and six months prior to his death. He was kept in bed for five days on the last occasion, but apart from these the patient had enjoyed fair health. He was a heavy drinker and smoker. The patient was married and had a large family. No family history of coronary artery disease was disclosed.

Autopsy was performed on 4.3.1945. The body was that of a well-built man. The colour of the face and the skin was pale.

The pericardial sac was distended with 300c.c. of dark fluid blood. The heart presented a recent, ragged, transverse rupture measuring $1\frac{3}{4}$ cm. by $\frac{1}{4}$ cm. situated /

situated about the middle of the anterior wall of the left ventricle and communicating with the pericardial sac. The rupture was surrounded by a haemorrhagic area. The anterior wall of the left ventricle was moderately thinned out especially towards the apex. The anterior descending branch of the left coronary artery was markedly sclerosed and occluded at a distance of 3 cm. from its opening by a recent red thrombus. The adjacent portion of the interventricular septum was not infarcted. The right coronary artery was moderately sclerosed but its lumen was patent throughout. The valves were normal. The aorta was atheromatous. The heart weighed 390 gm. The rest of the organs were fairly normal.

Case II

M.Y., a male, Greek, single, aged 66 years, retired, fell off his horse one morning on returning to his residence. He soon expired - no longer than a few minutes. It was thought he had sustained fractured ribs. He was seen leaning over the horse on his way back to his home.

The deceased was in the habit of riding for a number of years since he lived permanently in the country. His doctor gave an account of one moderately severe attack of coronary thrombosis nearly 18 months previously. He also added that he had had anginal pains over the last four years and that his blood /

blood pressure was in the neighbourhood of 160/110. The deceased was a teetotaler and smoked in moderation. It was also stated that his father died of coronary occlusion at the age of 65 years.

Necropsy on 11.5.46. revealed a well-developed muscular man. The face was pale. The pericardial sac contained 450c.c. of dark blood and clots. The anterior wall of the left ventricle presented a recent, ragged oblique rupture situated 6 cm. above the cardiac apex and to the left of the interventricular groove. It measured 1 cm. by $\frac{3}{4}$ cm. The state of the coronary arteries was similar to the previous case. A recent thrombus was found occluding the lumen of the anterior descending branch of the left coronary artery $3\frac{1}{2}$ cm. from its opening. The adjacent portion of the interventricular septum was not infarcted. The valves were normal. The aorta was markedly atheromatous. The heart weighed 430 gm. The lungs and liver were moderately congested.

D I S C U S S I O N

Spontaneous rupture of the heart was the direct result of acute coronary occlusion. The aetiology of acute coronary occlusion has already been discussed in detail. It only remains here to discuss primarily the direct factors in relation to rupture

and /

and its manifestations.

1. Incidence of Spontaneous Cardiac Rupture.

In 3,250 autopsies spontaneous cardiac rupture, resulting from acute myocardial infarction, occurred in two cases; an incidence of 0.06%.

The incidence of myocardial rupture in unhealed infarcts was 2.3%.

The incidence of sudden cardiac death due to such rupture was 0.6%.

The two cases were encountered in routine necropsy in the general Mortuary. None occurred among hospital patients.

2. Age Incidence .

The condition was encountered at 60 and 65 years of age. In view of the very limited number of cases in this series, it would be insignificant to correlate any relation between the age of the patients and incidence of rupture. Still this may suggest a tendency of rupture to occur after middle life, though perhaps in a longer series the condition may also be encountered before the age of 60 years.

3. Sex Incidence.

Both cases occurred in men. This may be in support of a tendency of rupture to occur in males on account of the labourious life of this sex. Such sex predominance will be noted to parallel the incidence of coronary sclerosis in men.

4. Race.

One case occurred in an Egyptian and the other in a Greek. This equal frequency in both races may

suggest, on the surface, a higher incidence of rupture among the Greeks, as these form only a small group of the total population of the country. Nevertheless, such ruptures were not encountered among other foreign nationalities residing in the country, and this does not signify that these were immune to cardiac rupture, though a number of them came to autopsies with acute coronary occlusion. It will, therefore, be an erroneous judgement in this connection to lay any stress on a racial factor.

5. Body Constitution and Associated Diseases.

The patients in both cases were well-built, and one of them was muscular and a sportsman. In both cases the hearts were hypertrophied. This suggests that a combination of over-weight and hypertension is of significance in predisposing to cardiac rupture consequent upon acute coronary occlusion. It may be noted that not necessarily the combination of these two factors must be present, and that a single factor, by itself, may thus be considered with gravity.

6. Social Conditions and Predisposing Factors.

The first case belonged to the hard working class of people, while the second one fell into the well-to-do class. However, irrespective of this wide gap between the two cases, as far as social conditions are concerned, rupture occurred in both. This suggests

one determining element combining the two cases, namely physical exertion. This was ascertained in the two cases. Had the infarction occurred at rest or, at least, under more amenable conditions, the likelihood of rupture may be reasonably lessened, though its outcome may be inevitable under all circumstances.

7. Mode of Death.

In both cases death ushered in very rapidly and its duration was between a few and fifteen minutes. It would be interesting to know how rapid would be the process of death if rupture had occurred at rest. However, it seems that ventricular rupture is immediately attended with death.

In both cases no symptoms seem to have attended the fatal issue.

8. Cause of Death.

In both cases death resulted from cardiac tamponade caused by a haemopericardium.

9. Pathological Findings.

In both cases acute coronary occlusion by a thrombus formation was the determining cause. Atherosclerosis of the coronary arteries was the basis.

10. Site of Occlusion.

Occlusion of the anterior descending branch of the left coronary artery occurred in both cases.

This /

This suggests that this branch is more commonly occluded than others, and that anterior ruptures are of a common occurrence. The thrombus was located at a distance of 3 cm. and $3\frac{1}{2}$ cm. in the two cases respectively.

11. Size of Rupture.

The ruptures were relatively of a small size, 1 to $1\frac{1}{2}$ cm. in length. No more than one rupture was encountered in one case.

C O M P A R A T I V E S T U D Y

1. Incidence of Cardiac Rupture.

According to Benson et al (1933), cardiac ruptures collected from literature until 1918 were 937 cases. For obvious reasons, however, the percentage of cardiac ruptures in hospital mortuaries is always low, because such patients usually die before they reach hospital. They cited Krumbhaar and Crowell (1925), who found 7 ruptures in 16,000 autopsies at a general hospital. They also referred to the Munich report of 7 in 13,000 as cited by Meyer (1888), and the Leipzig series of 9 in 8,000 (Krumbhaar.). De la Chapelle recorded 20 cases of rupture among 15,000 in the medical examiner's service in New York City. Beresford and Earl (1930 - 31) reported an incidence of 0.86% among hospital statistics. Benson et al gave /

gave an incidence of 0.57% and noted that most of their patients died without medical attention, 27 reaching the coroner's mortuary.

Edmondson and Hoxie (1942) found 72 instances of spontaneous rupture in 865 hearts which contained unhealed infarcts.

In this connection the two cases reported in this series almost proportionately coincide with the figure given in the Munich report.

In Benson et al series the incidence of cardiac rupture among the coroner's cases was 1.27%, but it should be noted here that their series also included examples of ruptures due to recent infarcts at the site of old cardiac aneurysms, syphilitic cardiac aneurysm, and dissecting aneurysm of a sinus of Valsalva. Such was not encountered in the present cases under discussion. However, in their series of 40 cases there were only 25 instances of rupture of the heart as a result of infarcts due to coronary occlusion. This lowers the incidence of cardiac rupture proportionately. Nevertheless the incidence of cardiac rupture in their private cases was 0.28%. Still, the last figure is higher 4.7 times than the one obtained in the present series.

Edmondson and Hoxie (1942) reported an incidence of 0.29% in a series of 25,000 autopsies between 1924 and 1941. They also pointed out that the incidence

of /

of myocardial infarction has been on the increase as they encountered in the first half of the autopsies 266 cases of myocardial infarction while 599 infarcts were found in the second half of the cases. Among 865 hearts which contained unhealed infarcts, 72 instances of spontaneous ruptures were found; an incidence of 8.2%. The corresponding incidence in the present series is 2.3%.

2. Age Incidence.

In Benson et al series of 25 cardiac ruptures due to acute coronary occlusion, the ages ranged between 50 and 86 years. Of these, five were between 50 and 59, nine between 60 and 69, eight between 70 and 79, and two above 80 years of age. This range indicates a greater incidence of rupture of almost equal figures in both the 6th and 7th decades of life.

Consistent with the above are Segal's (1945) figures reporting four cases with ages of 61, 61, 67 and 78.

Friedman and White (1944) gave an average age of 65.7 years, and Jetter and White (1944) reported an average age of 66.5 years.

The age incidence in the present two cases is in accordance with the above findings in general.

It was also noted in the series of Benson et al that the female cases were 56, 60, 73, 75 and 79 years

old /

old and in Segal's cases the females were 67 and 78 years old, a decidedly older age incidence in the female than in the male sex, which may suggest a milder course and perhaps a better prognosis of cardiac infarction in the female sex.

3. Sex Incidence.

Statistics give a predominance of rupture in the male sex. The predominance of the causes of atherosclerosis in the male sex is doubtless responsible.

Author	Year	Males per cent	Females per cent
Krumbhaar & Crowell	1925	58.1	41.9
Jetter & White	1944	62.5	37.5
Friedman & White	1944	70.0	30.0
Benson et al	1933	77.5	22.5

4. Social Conditions.

Among the series of Benson et al there were 3 labourers, 1 gardener, 1 truck driver, 1 farmer and 1 canner, namely about 30% of trades involving manual work. Others were 1 physician, 1 theatre manager, 1 conductor, 1 painter, and two retired. The first group in their series distinctly indicates that physical exertion is an immediate forerunner of ruptures and, as stated by them, death occurred in five cases following physical exertion, a factor stressed in the series under discussion. The female cases /

cases were all housewives.

5. Mode of Death and Size of Rupture.

In Benson et al series of 25 cases death was sudden in 16 cases and in 9 cases there was a history referable to previous coronary attacks.

In Segall's cases death occurred in one case within a half hour after the onset of fatal symptoms. The suddenness of symptoms and the very rapid death in these cases is in agreement with the present findings.

There have also been other cases on record in which death was instantaneous. Examples of these cases were given by Jetter and White (1944). It is interesting to note that death in some of these cases occurred at rest.

However, death may be delayed as described by Segall in a case of a man, aged 61 years, who died 96 hours after the onset of fatal symptoms. It is of interest that death after a double cardiac rupture may be delayed as long as 7 days. This is found in the case reported by Carrol and Cummings (1947) of a man, aged 60 years, whose heart showed a double rupture, one through the lower posterior surface of the left ventricle and the other at the base of the interventricular septum, measuring 4 cm. by 1.5 cm. and 3 cm. by 1.5 cm. respectively.

Other /

Other records of survival after rupture were recorded by the following observers:-

Freeman and Griffin (1932)	:	5½ hours.	(Hyman's case)
" " " "	:	17 hours	(Latham's first case)
" " " "	:	36 hours	
" " " "	:	38½ hours	
" " " "	:	72 hours	(Latham's second case)
Bayley and Fader (1941)	:	8 weeks.	
Moolten (1942)	:	4 months.	
Stanley (1937)	:	5 months.	
Gross and Schwartz (1936)	:	14 months.	

The following case is perhaps the longest survival on record. Wood and Livezey (1942) described a case of a male, aged 44 years, who had coronary occlusion at the age of 44 and perforation of the ventricular septum through the infarcted area. He lived four years and ten months and led a fairly active life despite congestive heart failure.

In connection with these ruptures, the general opinion held by all authors, is that rupture of the left ventricle after infarction is immediately fatal unless the area of softening is situated in the inter-ventricular septum. In the latter case death may be delayed several days, but prolonged survival is rare (Moolten, 1942 and Freeman and Griffin 1932).

That the diagnosis in such rupture may be missed is impressed by Freeman and Griffin, who stated that

the /

the symptoms may point towards an acute illness of some grave abdominal condition. However, finally symptoms attract attention to the heart. Perforation is ushered in by a renewal of syncope, a harsh systolic thrill, gradual extinction of the aortic second sound, progressive fall in blood pressure with obliteration of the pulse and pallor rather than cyanosis.

Occasionally myocardial rupture is attended with few or no symptoms, as in the cases under study. Symptomless ruptures were also recorded by Ellwood (1947), reporting on 4 cases; in two of them there was rupture of the left ventricle due to a recent infarction, yet clinically no symptoms presented themselves. In the third case, the patient was asymptomatic and refused to stay in bed. In the fourth case also, a rupture developed through the site of a new infarction.

6. Associated Diseases.

The question of hypertension has been stressed lately by Edmondson and Hoxie (1942). They found myocardial rupture in 25% of patients whose hearts weighed less than 400gm. and whose blood pressures were 140/90 mm.Hg. or above, after myocardial infarction. In 368 patients with heart weights of 400 gm. or over, and a blood pressure of less than

140/90 /

140/90 mm. Hg. after infarction, only 4% had cardiac rupture.

They also found that if old scarring was present in the myocardium the likelihood of rupture was one fourth as in unscarred hearts. The reasons given were that fibrous tissue in a scarred muscle was resistant to ischaemia, thus adding support to the necrotic muscle, and that in scarred hearts the collateral circulation, being greatly increased, lessened the liability of softening and of subsequent infarction.

7. Site of Rupture.

The general agreement is that the anterior wall of the left ventricle is the commonest site of rupture, as is noted in the present series.

Wearn (1923) out of 19 ruptures found 16 in this position.

Barnes and Whitten (1929) found the left ventricle involved in series of 47 ruptures. Among 36 of these ruptures, occlusion was in the left coronary artery, in 22 cases occlusion was in portions of the left ventricle driving its blood supply from the right coronary artery, and in 4 cases where the right ventricle was involved, there was also infarction of the left ventricle in the area of the right coronary supply.

Cowdry (1933) have seen one heart in which the

right /

right atrium was the sole seat of infarction in the heart.

Wagner (1929) stated that sometimes occlusion of the left coronary artery may be high enough to produce infarction of the papillary muscle leading to rupture of the muscle.

According to Freeman and Griffin (1932), cases of spontaneous rupture collected by E. Krumbhaar and Crowell showed the following distribution as tabulated:-

Site of Rupture	Total	Per cent
Right auricle	35	5.7
Left auricle	12	2.0
Right ventricle	63	10.2
Left ventricle	493	79.7
Miscellaneous	15	2.4

The miscellaneous group consisted mostly of ruptured papillary muscle, but also included two cases of rupture of the interventricular septum reported by Latham.

RUPTURE OF CARDIAC ANEURYSM

Historical Review:

The history of cardiac aneurysm dates back nearly 300 years.

Olus Borrich, as pointed out by Steel (1934), was among the earliest who reported on cardiac aneurysm in 1676. He reported on an aneurysm of the right auricle.

Delano Weihe (1944) referred to Pierre Dionis, who in 1696, reported on an aneurysm of the right ventricle.

Parkinson et al (1938) stated that the term cardiac aneurysm was used by Lancisius (1740), de Senac (1783) and Corvisart (1806) to signify a general enlargement of the heart cavities. They also referred to Galeati as having been the first to have given an account of aneurysm of the left ventricle in 1757, and that in the same year Hunter described an undoubted example, "At the apex it was forming itself into a kind of aneurism, becoming there very thin, that part was lined with a thrombus just the shape of the pouch in which it lay". In 1785, Walter described a specimen of apical aneurysm which he had received long before in 1759.

Crawford (1943), stated that Mathew Baillie in 1793, gave the first detailed description of an

aneurysm /

aneurysm as follows: "It sometimes happens, although I believe very rarely, that the heart becomes aneurysmal. This disease consists in a part of it being dilated into a pouch, which is commonly more or less filled with coagulated blood."

Ziegler in (1881), is credited with the introduction of the modern conception of ischaemic fibrosis, due to coronary sclerosis or thrombosis, as the pathological basis of the condition.

During the nineteenth century the condition became well recognised as a pathological rarity, and important papers were contributed by Breschet (1827), Thurnam (1836), and Legg (1883, 1844), who collected 90 cases observed subsequent to 1840 (Parkinson et al 1938).

Early in the twentieth century, Hall (1903) collected 112 cases of cardiac aneurysm and described them. Sternberg in 1914 made an exhaustive study of the subject and recognised four stages in the development of cardiac aneurysm:-

1. Anginal attacks.
2. Infarction and pericarditis.
3. Latent period.
4. Terminal stage with congestive failure or sudden death.

Case Material:

Rupture of cardiac aneurysm occurred in five cases. These are described below in order of occurrence.

Case 1.

A.M.S., a male, aged 50, a carpenter, while at work was noticed to have suddenly stopped his work, staggered, looked dazed for a few moments and then fell to the ground. He uttered no words, but gave a faint sigh and was noticed to have turned very pale. He was offered a drink of water but was unable to swallow. Smelling salts were of no avail, and he was pronounced dead when a first medical aid was called. Death was stated to have occurred within 30 minutes. No reliable history was obtained except for shortness of breath on exertion and loss of appetite for the last six months.

Autopsy was performed on 27.1.39. The body was that of a well-built, muscular person. Findings were as follows: The pericardial sac was distended with 300 c.c. of dark fluid blood. The heart was very pale and weighed 400gm. The ventricles, especially the left, were in a state of contraction. An aneurysmal dilatation of the left ventricle posteriorly, with a recent ragged rupture measuring 3cm. by 1.5cm. was found. The cavity of the heart was almost empty of blood. More than the distal half of the posterior interventricular branch of the right coronary artery was completely occluded, and evidence of old thrombosis was present. Sub-epicardial petechial haemorrhages, largest measured $\frac{1}{2}$ cm. across, were /

were scattered along the course of the posterior interventricular branch of the right coronary artery and its tributaries, as well as around the ruptured area. The left coronary artery was atheromatous, but its course was patent. The aneurysmal wall was thin, nearly 2mm. in thickness, and fibrous. The wall of the left ventricle was 1.75 cm. in thickness, and that of the right ventricle was 0.5cm. Evidence of old scarring of the wall of the left ventricle was present. The valves of the heart were normal. Atheroma of the aorta was present in a moderate degree.

Other findings, apart from the heart, were advanced cirrhosis of the liver, splenic enlargement, oesophageal varicies, moderate ascitis, haemorrhoids and bilharzial papillomata of the urinary bladder.

Case II

M.S., a male, aged 45, was found dead in his room. A history of breathlessness and cough for the last two years was obtained, but apart from this, he was able to carry on his work as a gardener. He was of a thin build, and it was stated that he had lost a good deal of weight in the last three months.

Necropsy on 22.1.42. revealed aneurysmal dilatation of the anterior wall of the left ventricle in its lower half. The wall of the aneurysm was 3 mm. thick. The pericardial sac contained 400c.c. of dark blood and a few clots. The heart weight was 400 gm., and the cardiac muscle was in a similar

state /

state to the previous case. The rupture of the aneurysmal wall was recent, and its edges were irregular and measured 4 cm. by 1 cm. The cavity of the heart contained about 5c.c. of blood. The anterior descending branch of the left coronary artery was completely occluded in its lower two-thirds, with petechial haemorrhages along its distribution. The right coronary artery was patent, but contained atheromatous patches. The wall of the left ventricle above the aneurysm was 2 cm. in thickness, the right ventricular wall was 0.5 gm. thick. The wall of the aneurysm was scarred. Old organised thrombosis of the occluded coronary artery was evident. No disease of the valves was found. The aorta presented a moderate degree of atheroma.

Other findings were:- Severe congestion and oedema of the lungs and a moderate degree of congestion of the liver.

Case III

M.M., a male, aged 50, heavily built, was found dead on the road. No history of illness, recent or past, was available. The nature of his work was uncertain, though it was stated that he had worked as a labourer for a number of years previously.

Autopsy was performed on 4.3.45. The pericardial sac contained 500 c.c. of dark fluid blood. The heart weighed 450 gm. The left ventricle presented a large aneurysm of the anterior wall in its lower half with a recent ragged rent, measuring

4 cm. /

4cm. by 1.5 cm. The wall of the aneurysm was 3 mm. thick. The cavity of the heart was quite empty. The anterior descending branch of the left coronary artery was partly patent in its proximal third, the distal two-thirds were completely occluded with evidence of thrombosis. The right coronary artery was patent throughout, but presented few atheromatous plaques. No significant ecchymosis were found in the cardiac muscle, or along the course of the left coronary artery. The wall of the left ventricle above the aneurysm was 2.25 cm. thick, the right ventricular wall was 0.5 cm. thick. The valves were normal.

The lungs were congested and oedematous. The liver and spleen were congested. The kidneys showed arteriosclerosis.

Case IV

M.L., a male, aged 45, jew, died suddenly in a cafe. He was talking to a friend and was noticed to have bent over the table and become very pale. An unsuccessful attempt was made to rouse him. He was immediately carried to the nearest chemist, but was declared dead. Death was stated to have occurred in less than two minutes. The history was that of a heaviness in the chest, sometimes amounting to pain, occurring especially after meals, and dyspnoea on exertion for the last two or three years. No disabling symptoms were complained of lately. He was employed as a salesman.

Post mortem was done on 21.2.46. The body was that of a very obese person. The pericardial sac was /

was full of dark fluid blood. The heart weighed 530 gms., and its muscle was completely relaxed. The upper half of the right ventricle anteriorly presented an aneurysmal dilatation of its wall, 1.5 mm. thick, with a recent irregular rent measuring 3.5 cm. by 1.5 cm. The main right coronary artery was nearly completely occluded. The left coronary artery was patent and showed few atheromatous plaques. The wall of the left ventricle was 2.5 cm. thick. No ecchymoses were found in the cardiac muscle. The aneurysmal wall was scarred.

The lungs were pale, the liver and spleen were slightly enlarged and congested, and the kidneys were moderately arteriosclerotic.

Case V

F.I.A., a male, aged 70, Sudanese, died suddenly at his home shortly after he had taken a bath. He collapsed while he was having a cup of tea, was seen to have dropped the cup, and for about a minute he tried to hold on to his seat, but eventually died within two minutes from collapse.

The history was that, previous to the last ten years he was employed as a door-keeper for most of his life. During this term of employment, he had about three or four severe pains in the chest, which were attributed to weakness of the heart. He received medical attendance on these occasions and was able to resume his work shortly after his attacks. During the last ten years he was not at work, though enjoyed good health. In the last two days before death, he complained of pain in the legs.

Autopsy was done on 6.3.46. The body was that

of /

of a very thin person. The pericardial sac contained clotted blood which weighed 300 gm. The anterior wall of the left ventricle presented an aneurysmal dilatation on its lower half, 3 mm. thick, with a recent ragged rupture, 3 cm. by 0.2 cm. The heart weighed 380 gm. The state of the myocardium and coronary arteries was similar to that in Case III. The aorta was calcareous. The valves of the heart were normal.

The lungs were congested and oedematous. There was no evidence of lung infarction. No vascular lesions were found in the legs.

DISCUSSION

Rupture of cardiac aneurysm accounted for 1.4% of sudden cardiac death. This relatively small proportion points to the infrequent occurrence of ruptured cardiac aneurysm, although, does not exclude its occurrence in a proportion of other cases without rupture. Nevertheless, no unruptured cardiac aneurysms were encountered in autopsies carried out over a period of nine years. However, one may assume that not all cardiac aneurysms eventually rupture and that a certain degree of longevity with cardiac aneurysm may be expected.

1. Incidence of Cardiac Aneurysm.

Five cases of cardiac aneurysm were encountered among 3,250 autopsies. This gives an incidence of 0.15%.

Incidence of cardiac aneurysm in old standing coronary artery occlusion was 1.7%.

2. Age Incidence.

The age incidence was between 45 and 70 years. This is a wide range and may suggest that a reasonable term of longevity, as referred to before, may be compatible with this condition.

The average age group in this series was 52 years.

The incidence was common about middle life and was divided equally between the ages of 45 and 50. It was rare in old age; this may be accounted for by death occurring either at an earlier time from such conditions as coronary artery disease before it had progressed to myocardial scarring and aneurysmal dilatation, or from an aneurysmal cardiac rupture at an earlier date.

It is noted in this series that cardiac aneurysm was not encountered before 45 years of age.

3. Sex Incidence.

All cases occurred in males. This may be considered as evidence of a greater incidence of the condition in this sex. It is also consistent with the frequent /

frequent incidence of coronary atherosclerosis in males.

4. Race.

That the condition occurred three times out of five among Egyptians is accounted for by the majority of the population. However, race seems no bar as it also occurred, though only once, in a Sudaneze and in a Jew.

5. Site of Aneurysm.

Cardiac aneurysms occurred four times as frequently in the wall of the left ventricle as in the wall of the right ventricle, and three times as frequently on the anterior wall of the left ventricle as on the posterior wall.

All aneurysms of the wall of the left ventricle occurred in its lower half and also involved the apex.

The only incidence of aneurysm of the right ventricle occurred on the upper portion of its anterior wall.

The incidence of aneurysm of the left ventricle is 80%.

So far, in this series of total autopsies, neither interventricular septal aneurysm, nor aneurysms of the wall of the auricles were encountered.

6. Size and Number of Ruptures.

The ruptures measured between 3 cm. to 4 cm. in
length /

length. No more than one rupture was found in any one case.

7. State of the Heart and Associated Diseases.

In all cases the heart was enlarged, ranging from 380 gm. to 530 gm.

In two cases there was arteriosclerosis of the kidneys. In one case there was cirrhosis of the liver.

8. Symptoms, Mode of onset and Duration.

In two cases no history was available. In the remaining three cases, no prodromal symptoms occurred, and death has ushered in with collapse.

In one case there was a complaint of pain in the legs for two days before death. Whether such pain was of a spasmodic origin, or a peripheral type of cardiac pain, it is difficult to assess. However, no pathological lesions were encountered in the arteries of the legs.

In the three cases where a history was available, rupture occurred with a dramatic onset and manifested itself by a severe collapse. The process of death was almost instantaneous in two cases, and occurred within half an hour in the third case. The rapidity of death is accounted for by the large size of the rupture and the rapidly developing tamponade. Perhaps, in other instances where the size of the rupture is small, or of a valvular type, prodromal symptoms

may /

may appear and cardiac tamponade is delayed. Death in any case would be inevitable.

9. Previous History.

Dyspnoea was complained of in 4 cases and heaviness or pain in the chest, especially after meals, in one case. Heaviness or pain in the chest after meals is explained obviously by pressure of the distended stomach, with food, on the heart.

The history of Case V. definitely points to a previous, recurrent, coronary artery occlusion of some years standing, with postmortem evidence of old thrombosis of the anterior descending branch of the left coronary artery.

10. Body Condition.

In two of the cases the body weight was on the heavy side, in one case definitely obese, and in the remaining two cases the body was thin. This may show a preponderance of the condition to occur in fat people.

11. Social Conditions.

All cases in this series belonged to the working class.

12. Predisposing External Factors.

In all five cases no direct external factors, normally predisposing to rupture, were encountered with the exception, perhaps, of the first case

(carpenter)

(carpenter), who died while engaged in his normal manual work.

13. Pathological Findings.

In all five cases severe atherosclerosis of the coronary arteries was seen. The coronary vessel supplying the site of aneurysm was occluded and showed evidence of old organised thrombosis. The aneurysmal wall consisted of scarred fibrous tissue.

14. Mechanism of Death.

The mechanism of death in all cases was due to cardiac tamponade produced by the haemopericardium resulting from the rupture of the thin aneurysmal wall.

C O M P A R A T I V E S T U D Y

1. Incidence of Cardiac Aneurysm.

Parkinson et al (1938), combining the incidence of cardiac aneurysm in autopsies of various observers, found that it occurred in 9% of cases of cardiac infarction.

Ball (1938), quoted Appelebaum and Nicolson (1935) who, in a study of coronary artery disease, found 57 cases of aneurysm of the heart out of 150 cases in an atherosclerotic group; an incidence of 38%.

This is rather an outstanding proportion which would

make /

make one very suspicious of cardiac aneurysm occurring in a large group of patients suffering from disease of the coronary artery. However, Ball himself, looks upon cardiac aneurysms as rare and an unusual pathological condition. In this present series, incidence of cardiac aneurysm was far below any figure given above. The incidence of the condition in all autopsies was 0.15% and among the atherosclerotic group was 1.7%.

2. Age Incidence.

Burn et al (1943) reported a cardiac aneurysm in a girl thirteen years old. The origin of the aneurysm was sharply limited to the fibrous connective tissue of the mitral valve and only involved the muscular wall of the left ventricle on the posterior portion. The coronary vessels were thin walled and patent throughout. It is believed that the anatomical characteristics of the structure suggests that it was a congenital abnormality arising from a fibrous band of the mitral ring. Burn states that he could find only three other reports in literature of cardiac aneurysm similar to the one described by him. Such a congenital, rare anomaly was not found in the present series. However, the interesting point in this case is the relatively very young age of the patient who, as described by Burn, was seized by a sudden illness while at play and died within a

short /

short time before any medical aid could be obtained.

Hunter and Benson (1933), referred to the only other report in literature that described a similar cardiac deformity.

Shookhoff and Douglas, in a series of 145 cases, reported that the average age of patients was 57.8 years, with only three patients under the age of 40. They also reported on a case of their own who was only 35 years of age.

Parkinson et al (1938) reported on two cases in males aged 30 and 39, and one in a female aged 42. The oldest case reported by them was of a man aged 74. The average in their series of fifteen cases was 55.7.

This, in general, is consistent with my findings that the condition is commoner in about middle life, though, the average age of the patients in both series ranges between 3.7 to 5.8 years higher than the average age found in my cases.

3. Sex Incidence.

Steel (1934) pointed out that about 80% of cardiac aneurysms occur in males.

In the series of Parkinson et al (1938), 93.4% of cardiac aneurysms occurred in males.

This is in accordance with my findings, though all my cases were males. Perhaps, in a larger series, an incidence in the female sex would have been

encountered /

encountered. However, the preponderance in this series of the male sex is quite evident, and this is again undoubtedly in accordance with the preponderance of atherosclerosis in the male sex.

4. Race:

No definite racial factor is described, apart from the fact that Hewbrews are especially prone to disease of the coronary arteries, White (1946). In this connection although a heavy incidence of coronary sclerosis is noted among the Egyptians, yet the incidence of cardiac aneurysm is proportionately very small. It would, therefore, be very difficult in view of this fact to accept race as an aetiological factor. However, in argument against the latter statement, death from coronary artery disease at an earlier time of life, before the disease had progressed to myocardial scarring and aneurysmal dilatation, may be taken into consideration.

5. Site of Aneurysm:

(a) The Ventricles:

According to Hall (1903), in a collection of 112 cases, the various sites of aneurysm are tabulated as follows:-

SITE /

SITE OF CARDIAC ANEURYSM

SITE	NO. OF CASES
Left Auricle	92
Right Ventricle	1
Left Auricle	2
Ventricular Septum	
Muscular part	8
Membraneous part	7
Auricular System	2

Hall again in his collection found that 76% of cases occurred at or near the apex of the heart and 19 were situated in other parts of the ventricle. He also recognised in a collection of 243 aneurysms of the left ventricle that fully three-fifths were situated at the apex.

Crawford (1943) emphasised that most aneurysms occur in the left ventricle since myocardial infarction of the right ventricle is uncommon. He gives the apex and its immediate vicinity as the most frequent site.

Ball (1938) quoted Appelebaum and Nicolson, who reported on 56 out of 57 cases involving the left ventricle. This gives an incidence of 98.2% in the left ventricle.

Schwedel and Gross (1939) in a review of 81 cases found cardiac aneurysms 48 times in the anterior or lateral portion of the left ventricle, 21 times in the posterior /

within an hour from rupture of a thin fibrous pericardial wall of an aneurysmal sac.

It is interesting to note that Martin found that the anterior descending branch of the left coronary artery had no connection with the aneurysm.

He presumes that the sac in this anatomic position is developmental in origin, and that on account of its anatomic position is not likely to produce symptoms.

He gave the causes of intramural aneurysms as follows:-

- (i) Herniation of the endocardium due to developmental myocardial defect.
- (ii) Endocardial herniation into a localised area of myocardial fibrosis resulting from antenatal trauma, infection or localised coronary occlusion.
- (iii) True coronary aneurysm.
- (iv) Solitary abscess.

He also referred to Vaquez (1921) and Joachim and Mays (1926-1927), who described two similar cases, though in their cases a very clear-cut history of trauma was obtained.

6. Congenital Aneurysms.

As stated by Crawford (1943), these have been reported in rare instances and mainly involved the pars membranacea.

7. Multiple Cardiac Aneurysms.

Though /

Though the majority of cardiac aneurysms are single, multiple aneurysms have been reported on the following occasions. Delano and Weihe (1944) referred to Thurman who in 1838 reported a case in which 4 aneurysms were found in the left ventricle. They also quoted Fujinami who, in 1900 reported a case with three aneurysms, one at the apex, one in the posterior wall below the mitral orifice, and one of the septum.

Cookson (1929) reported on a female, aged 43, with three aneurysms occupying the posterior portion of the left ventricle.

Hall (1903) referred to Kundrat, who in one case found three distinct aneurysms. Hall, however, stated that cardiac aneurysms are nearly always single. In a series of 92 cases, he found that 83 were single.

In the present series all were single.

8. Duration of Life after Diagnosis:

Crawford (1943) reported no ruptures in one series of his cases. Young and Schwedel (1944) gave ten years survival period in a patient, aged 48, who was observed during nine years before his death with progressive enlargement of an aneurysm of the anterior wall of the left ventricle and the greater portion of the interventricular septum. At post-mortem the aneurysm measured 10cm. by 8cm. by 7cm. Its wall was 1mm. to 3mm. in thickness; its consistency was

cartilage /

cartilage and calcified deposits.

Parkinson et al (1938), in a series of 15 cases, reported on a range of longevity after the diagnosis of ventricular aneurysm, as from one week to thirty months, averaging 10 months.

Dressler and Pfeiffer (1940), reporting on 10 cases, gave the duration of life following recognition of aneurysm as from three weeks to seven years. They even stated that those who achieved the upper ranges were able to carry on normal activity or even strenuous physical effort. Wilson (1919) reported on a case of a female, aged 60, with a cardiac aneurysm, who was quite active and thought nothing of walking twenty miles. However, twenty-four hours before she died she walked one mile to the Church.

Fulton (1941) reported an apical aneurysm in a patient, who was still alive one and a half years after the recognition of the lesion, and in another case, a large posterior aneurysm with a probable duration of five years from its recognition.

Gross and Schwedel (1941), in a post-mortem analysis of cases of cardiac aneurysm gave the opinion that a ten years survival period was most unusual.

Penner and Peter (1946) reported on a 43 years old infantry officer, who had his first attack of coronary occlusion in 1929. He was working in an administrative /

administrative capacity from 1924 to 1944. In 1934 he was active in building and sailing and deep-sea fishing. This is a long term, about fifteen years of active life, after recognition of the condition.

In my own cases, no clinical history was obtained suggestive of the recognition of the condition during life. However, it may be, that in the last case, the aneurysm was of some years duration, considering the patient's history.

9. Pathology and Method of Formation of Cardiac Aneurysm.

It has been agreed by various observers that a cardiac aneurysm results from a dilatation of a myocardial scar, an event which is late in development, and which occurs after months or even years from cardiac infarction (Boyd 1944).

Crawford (1943) quoted Sternberg (1914), who stated that in 84.8% of the 207 cases which he analysed, cardiac aneurysms followed coronary occlusion. This observation is in accordance with my own series.

Parkinson et al (1938) gave the following aetiological varieties.

1. Arteriosclerotic.
2. Syphilitic.
3. Mycotic.
4. Rheumatic.
5. Congenital.
6. Traumatic.

That /

That syphilis plays a very minor role in the development of cardiac aneurysm is confirmed by Braunstein et al (1940), who reported that the total number of leutic cardiac aneurysms was 19 cases. They described the nineteenth case which was of a thirty year old negress.

Steel (1934), summarising the anatomical structures involved in cardiac aneurysms, displayed that the left coronary artery is usually longer than the right, and that a network of anastomosis is widely distributed not only between the right and left coronary arteries in their capillary and pericapillary branching, but also between coronary arteries and vessels of adjacent and attached organs and between branches of each coronary artery. These anastomoses involve the fine peripheral branches in the subendocardial and subepicardial layers, and thus it is clear that there is no anatomic end artery in the heart.

Sutton and Davis (1931) studied the effect of exercise on experimental cardiac infarction in dogs. Their experiments threw a considerable light on the formation of cardiac aneurysms.

The experiments were formed on the following basis:-

1. A preliminary training period of five days for dogs on a motor driven treadmill at a rate of over three miles was given, after

which /

which the dogs were subjected to ligation of the anterior descending branch of the left coronary artery 1 cm. to 2 cm. from its origin.

2. (a) One dog was given a six-day post-operative rest interval.

(b) Two dogs were given a two-day post-operative rest interval.

(c) Two dogs were given a one-day post-operative rest interval.

3. Exercise was then resumed after the intervals of rest stated and was continued for periods varying between 70 and 309 days, when each animal was autopsied.

Autopsies revealed the following:-

(a) dog developed a small puckered fibrotic scar without thinning of the ventricular wall.

(b) One of the dogs in this group developed a well defined aneurysmal sac. The anterior descending ramus of the left coronary artery was completely occluded. The other dog showed thinning of the apex of the heart which was reduced to 2mm. to 3 mm. in thickness by replacement fibrosis.

(c) One dog in this group showed defined

pouching /

pouching of the endocardium in a defined area of fibrosis near the apex.

The relation of effort in predisposing to cardiac aneurysm as a sequence to coronary occlusion is quite evident from the above experiments. Effort involves over-taxation of the ischaemic cardiac muscle, and hence formation of cardiac aneurysm, as a complication of coronary occlusion, can be prevented if adequate rest is given to the myocardium for a long period after the episode of coronary occlusion.

In this connection it has been stated by Delano and Weihe (1944) that cardiac aneurysms may be found as early as six weeks after a coronary occlusion. It is interesting to note that Shookhoff and Douglas (1931) reported cardiac aneurysms appearing within a week or two of coronary occlusion.

It should also be noted, as pointed out by Ball (1938), that a pre-existing hypertension is naturally an added factor in enhancing formation of cardiac aneurysms.

10. Time between Rupture and Death.

Martin (1946) reported death within one hour from rupture of an intramural cardiac aneurysm in a male aged 20.

Parkinson et al (1938) found that in a group of 7 patients with cardiac aneurysms, 2 died suddenly,

4 from /

4 from congestive failure and 1 from cerebral embolism.

In my own cases death occurred within two to thirty minutes of rupture.

In every case the pericardial sac was found to be distended with blood and it can, therefore, be suggested that pericardial tamponade was the immediate cause of death.

11. Symptoms referable to Cardiac Aneurysms.

Ball (1938), stated that aneurysms of the posterior wall of the left ventricle, as yet, have not been recognised clinically.

Crawford (1943), stated that there are no symptoms of aneurysms of the heart. They may be asymptomatic, but as a rule symptoms due to congestive heart failure are present. Sometimes angina pectoris is the only complaint.

Parkinson et al (1938) in a series of 15 cases encountered the following symptoms:- Anginal pains, palpitation, dyspepsia and epigastric pain. In one case of a female, aged 42, complaint was palpitation of two weeks duration. She, however, died of broncho-pneumonia.

Analysis of these symptoms and others in my own cases points to the fact that in some cases of cardiac aneurysm, there may be few symptoms bearing directly on the condition, while in other cases symptoms are not related to the cardiac state.

A D I P O S I T Y O F T H E H E A R T

Case Material:

Adiposity of the heart was attended with myocardial rupture on two occasions.

Case I.

M.S.E., a male, aged 60, a railway porter was seized while at work with a severe precordial pain immediately followed by collapse. A doctor examined him ten minutes later and found him dead. It was stated that the deceased was carrying two heavy trunks, one on his back and the other held by his hand when he developed his fatal symptoms. On the morning of his death he was not especially engaged in much heavy work previous to his fatal issue and nothing unusual was noticed about him on that particular day. However, it was stated that the deceased had been breathless for the last four years. His wife stated that he was a moderate eater but a heavy drinker and smoker, though of late he had been very moderate with his drinks on account of his breathlessness. A history of obesity in the family on the mother's side was obtained although it was stated that his mother was a sufferer from elephantiasis.

Autopsy was performed on 8.4.40. The body was that of a very obese person. The face, lips and nails were deeply cyanosed. The pericardial sac was distended with 450c.c. of blood, mostly fluid. Situated on the anterior wall of the right ventricle, near the apex, there was a recent, ragged rupture measuring $2\frac{1}{2}$ cm. by 1cm., communicating the ventricular cavity with the pericardial sac. The left ventricle was markedly dilated. The heart was of a yellowish /

yellowish/brown colour . It weighed 400gm. The subepicardial fat was markedly increased and was found to have completely replaced the wall of the right ventricle and also appeared under the epicardium. A few dispersed atrophic muscle fibres were also seen. The lateral wall of the left ventricle presented a moderate degree of adiposity. The coronary arteries and their branches were very tortuous and embedded in fatty tissue. Their lumina were patent. The right coronary artery presented a moderate degree of atheroma. The valves were normal and the aorta presented few atheromatous plaques.

The lungs were congested and oedematous. The liver and spleen were moderately enlarged and congested. The kidneys were reduced in size but otherwise of a normal structure. The bladder presented bilharzial papillomata. The right ureter was slightly thickened and dilated and presented at its lower end few sandy patches. The brain and meninges were normal.

Case II.

A.M., a male, aged 60, single, a quarry foreman, while helping one of his men to unload a heavy cart by tilting it to one side, he was seized with severe pain in the chest and became very dyspnoeic. He was helped for no more than ten yards when he suddenly fell. He perspired a great deal and it was thought he had exhausted himself with the effect of the hot weather. He was given a drink of water which he was able to swallow with some difficulty. He was able, with some effort a few minutes

later /

Case II Cont'd.

later to utter a few words asking his men to take him home but he died thirty minutes after the commencement of symptoms.

It was stated that he had been a heavy smoker and occasionally indulged in drinking. His state of health lately was unsatisfactory and he was absent from work four times in the previous two months on account of "heart trouble", although no medical treatment was sought. No family history was available to ascertain a tendency to obesity.

Necropsy on 6.6.47. The body was that of a very obese individual. The pericardial sac was fully distended with 600c.c. of fluid and clotted blood.

On the lateral wall of the right ventricle about the middle and running partially on the anterior wall, a recent, irregular complete rupture of the ventricular wall, measuring 3cm. by $\frac{3}{4}$ cm. was found. The wall of the right ventricle was completely relaxed and the left ventricle was moderately dilated.

The whole thickness of the wall of the right ventricle was completely replaced by adipose tissue, and hardly any scattered atrophic muscle fibres were left.

The coronary arteries were similar to the previous case, as was also the state of the valves and the aorta. The heart weighed 390gm.

The lungs were congested and oedematous. The liver was enlarged and congested and the spleen was congested /

congested. The rest of the organs, including the cranial contents presented no pathological findings.

N.B. Discussion of these two cases is included in Chapter III on Adiposity of the Heart.

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CHAPTER III

ADIPOSITIVITY OF THE HEART

ADIPOSITY OF THE HEART

Historical Review:

This condition is hardly discussed at any length now-a-days in modern text books of medicine and pathology though it received a tremendous attention at one time by older writers, and many cases of sudden death were attributed to this cause.

Boyd (1943), described this condition, under the term of fatty infiltration, as a "lipomatosis of the heart, with deposits of fat under the pericardium and in the interstitial tissue". He considers it merely part of a general adiposity, and that when it is very marked there may be interference with the heart's action.

"In the past, two distinct conditions have commonly been confused, and both have been alluded to as fatty heart. These are, (1) the state in which there is an abnormal increase in the amount of fat in the subepicardial connective tissue and in which penetration or infiltration of fat into the connective tissue lying between the muscle bundles and the muscle fibres takes place and, (2) the state in which fatty changes take place within the cell (cytoplasm) and which most pathologists, (Mallory 1914), believe to be the result of a diminished utilization (oxidation) of the fat normally brought to the muscle cell

(Smith /

(Smith and Willius, 1933)".

"Many observers now agree with the views advanced by Rosenfeld (1901) and Herxheimer (1901) that the fatty change that occurs within the cytoplasm of the cell, so-called fatty degeneration, is in reality an infiltrative process rather than true degeneration and is probably the result of two causes: a disturbance of cellular nutrition and toxaemia", (Smith and Willius, 1933).

Smith and Willius also stated that an increase in the amount of fat in the subepicardial connective tissue is best designated, as pointed out by Beattie and Dickson (1948), as adiposity of the heart.

William Harvey, according to Pratt (1904), published the first observation on fatty heart in the 17th century (Smith and Willius 1933). Harvey reported on the body of a very corpulent man, describing the heart as being completely covered with fat (cor adipe plane tectum). At necropsy of the body of Parr, he found the heart large, thick, and fibrous, and containing a considerable quantity of adhering fat, both in the circumference and over the septum, but he did not attribute the patient's death to this cause.

Corvisart, in 1818, according to Saphir and Corrigan (1933), admitted the possibility of fatalities due to fatty degeneration, but had never observed any.

He /

He cited three fatal cases described by Kerckring, Bonnet and Morgagni. Leyden, in 1882, stated that death in some patients can be explained by (fatty heart). A case of historic interest is the one described by John Cheyne in 1818, in which the peculiar type of respiration now known as Cheyne-Stokes respiration, was described. At that time, Cheyne-Stokes breathing was thought to be almost pathognomonic of fatty heart, (Smith and Willius 1933).

Hamilton, in 1889, considered that atrophy of the muscle fibres associated with fatty infiltration precedes the infiltration instead of being the result of mechanical pressure of the fat on the muscle. He referred to cases recorded by Fothergill and by Leyden in which fat had accumulated to the extent of interfering with the motion of the heart.

Broudel, in 1895, stated that a patient may die unexpectedly as a result of this condition. Pepper, in 1896, stated that in cases of considerable deposit of fat, the fibres undergo atrophy and degenerative changes from pressure. The myocardium may be reduced to a narrow rim. The cavity is dilated, and rupture of the heart is not uncommon. He considered a hereditary tendency, overeating, excessive drinking of alcoholic beverages and sedentary habits as powerful determining factors. Rosenbach, in 1899,
mentioned /

mentioned that a nutritional disturbance causes muscular atrophy of the heart and secondary replacement by fat. Fisher, in 1903, opposed the idea of cardiac weakness due to fatty infiltration, and stated, "The fat going between the muscle fibres may occasionally be very great in amount, yet the muscle fibres prove on microscopic examination to be quite healthy". Wegelin in 1913, stated that fatty infiltration of the myocardium may cause sudden death. Eyselen, in 1914, mentioned two patients with marked fatty infiltration of the myocardium who died suddenly. Aschoff, in 1919, stated that fatty infiltration of the myocardium per se seems less likely to be the cause of insufficiency of the heart. Henry and Smith, in 1919, held that fatty infiltration occurs frequently and that its presence should be suspected during life in patients with cardiac disease who exhibit a general tendency to obesity. Kratter, in 1921, stated that a fatty heart may lead to sudden death, especially on occasions of muscular activity, overfilling of the stomach, psychic excitement and defecation. A fatty heart is found in almost all drunkards. Kaufmann, in 1922, distinguished between fatty degeneration and fatty infiltration of the myocardium, and stated that fatty infiltration may lead to unexpected death on occasions demanding increased cardiac activity, and that rupture of the heart may occur if the myocardium is atrophic or the

seat of fatty degeneration. Vagnet, in 1924, considered that uncomplicated fatty infiltration cannot cause death. However, rupture of the myocardium, in presence of myocardial ischaemia by sclerosis, is prone to occur with fatty infiltration of the heart. Vagnet also quoted Gallaverdin, who distinguished three forms of fatty infiltration of the heart, a benign form with indefinite symptoms, an intermediate form with cardiac asthenia, and a severe form with angina pectoris, acute oedema of the lungs, crises of heart failure or of uraemia. Mönckeberg, in 1924, referred to fatty infiltration of the conducting system. Karsner, in 1926, considered the possibility of disturbance of the function of the heart in excessive fatty infiltration of the myocardium. Norris and Landis, in 1929, stated that dyspnoea is not uncommon with fatty infiltration of the heart. Larday, in 1930, remarked that an examination of 160 adipose persons revealed, in 90 per cent, objective disturbances of the circulation as found by determination of the pulse rate, arterial and venous pressure and vital capacity. White, in 1946, stated that fatty infiltration of the myocardium, especially involving the right ventricle, is a definite entity, most common in middle-aged and elderly women. In rare cases this condition has been blamed as the primary or secondary cause of heart failure.

C A S E M A T E R I A L

Adiposity of the heart was encountered in six cases. Two were responsible for cardiac rupture and sudden death and four resulted in sudden death without rupture. The first group of two cases has already been described in Chapter II on Myocardial Rupture. The second group of cases is presented as follows:-

Case I.

H.S.S., a male, aged 60, a cabman, single, was found dead in the stable about six o'clock in the morning. He was seen alive last about midnight when he had unsaddled the horses and was stated to have chatted to a fellow friend for a few minutes. His friend noticed that he was under the influence of alcohol. The body was discovered, fully dressed, in bed lying on its back. A statement to the effect that the deceased was a habitual drunkard was agreed upon. No history of recent or past illness was available. A history of obesity in the family was denied.

Necropsy, 6.3.40. The body was that of an obese individual. The face, mucous membranes and the nails were cyanosed.

The heart was of a pale yellowish colour, very flabby, and the right ventricle was completely relaxed. It weighed 340 gm. The epicardium was smooth and glistening.

A marked increase of the subepicardial fat was present. The fat was found to have extended into

the /

the myocardium of the right ventricle, and replaced most of its muscle fibres. The remaining fibres were atrophied. A small amount of fat had also extended into the interventricular septum.

The coronary arteries showed few atheromatous plaques, and were patent throughout their course, though quite tortuous.

The valves were normal. The aorta presented a moderate degree of atheroma, but its wall was reasonably elastic.

The lungs were congested and slightly oedematous. The liver was enlarged and congested. The spleen was also congested. The kidneys were normal. The stomach was empty of food, and the mucous membrane was slightly hyperaemic. The brain and meninges were normal.

A sample of blood and of cerebrospinal fluid were tested for alcohol. The results proved the presence of alcohol in both, in a concentration of 80mgm.% and 90mgm.% respectively.

Case II.

F.O.W., a female, aged 50, a woman of uncertain character, collapsed suddenly in a public bar, after a short complaint of severe precordial pain and dyspnoea, not exceeding 45 minutes. She was rested on a couch but still her symptoms continued and even grew more severe. A doctor who examined her fifteen minutes later stated that she was very dyspnoeic, her pulse rate was 90 and of a poor volume. The blood pressure was 100/60 and the heart sounds

were /

were weak. The chest was clear. He gave her a morphia injection. He was of the opinion that she had a coronary occlusion. The patient's condition became increasingly worse, the dyspnoea becoming more severe and finally she died in a minor fit of convulsions. It was stated that the deceased was a habitual drunkard and on the night of her death had partaken of a large, late supper with some friends and had drunk four glasses of whisky. It was also stated that up to the onset of her fatal symptoms she was in apparently normal health. Past history was irrelevant, although one witness gave evidence that the deceased once or twice had complained of a fainting attack and dimness of vision. No family history was available.

Autopsy: 9.10.40. The body was that of a well-nourished person. A moderate degree of ankle oedema was present.

The heart was pale brownish, flabby and dilated. It weighed 280 gm.

The subepicardial fat was excessive in amount. The right ventricle was almost completely replaced by a fatty tissue. The interventricular septum, and the lateral wall of the left ventricle were invaded partly by fat. The coronary arteries were patent throughout. Few atheromatous plaques were found in the left coronary artery. The valves were normal, though few atheromatous plaques were seen on the left posterior cusp of the aortic valve. The aorta presented few atheromatous plaques.

Examination of the abdominal and cranial contents revealed no pathological condition that might account

for /

for her death. The liver and spleen were markedly congested.

A sample of blood was tested for alcohol. It revealed its presence in a concentration of 100mg%.

Case III.

B.S., a female, aged 65, a Sudaneze, widow, was found dead in bed. It was stated that she had attended a Sudaneze party "Zaar", and that she involved herself in a lot of native dances and drank a good deal of "booza", a national drink. She also had plenty of food. It was added that she was a frequent visitor to these special parties, and that she had always enjoyed herself and never left before the end of the parties, taking part in many dances without showing tiredness or fatigue. The deceased had two daughters - both obese.

Necropsy: 3.8.45. The body was that of an obese person.

The heart was yellowish brown in colour, flabby and dilated. It weighed 420 gm.

The epicardium was smooth and glistening.

The subepicardial fat was markedly increased. The right ventricle was largely replaced by fat which appeared also under the endocardium. The coronary system was patent throughout.

The aorta presented very few streaks of atheroma.

The lungs were congested and slightly oedematous. The liver weighed 1220 gm. and was congested. The spleen was also congested. The kidneys were normal. The stomach contained undigested food. An ovarian cyst /

cyst, the size of a large orange was present on the left side. The brain and meninges were normal.

A blood specimen contained 65mg.% alcohol.

Case IV.

E.M.R., a female, Italian, aged 60, died suddenly on a motor car journey, complaining of a severe dyspnoea lasting for about 90 minutes. The dyspnoea had started after 30 minutes on the journey and the family had to return. The patient was stated to have had during the last two years occasional attacks of dyspnoea on effort and also suffered from dyspnoea after late meals, but otherwise was in a fair state of health. A past history was given of what seemed to have been pneumonia 35 years ago. The patient ten days previous to her death had two teeth extracted. Death occurred before any medical attendance was available. A history of obesity in the family on the mother's side was obtained.

Autopsy: 6.12.45. The body was that of a well-nourished individual. The heart was pale brown, very flabby and dilated. It weighed 330 gm.

The epicardium was smooth.

The subepicardial fat was markedly increased. The myocardium of the right ventricle was largely replaced by fat, and the remaining muscle fibres were atrophic. The coronary system was patent throughout, though few atheromatous plaques were found in the course of the main vessels, especially the right coronary artery. The aorta presented a severe degree of atheroma. The valves were normal.

The lungs were congested and oedematous. The liver and spleen were normal in size but congested.

The /

The abdominal and cranial contents were found normal.

Examination of the buccal cavity revealed clean sockets of the extracted teeth.

DISCUSSION

1. Incidence of Adiposity of the Heart.

The condition was encountered 6 times in 3250 autopsies, an incidence of 0.18%.

2. Age Incidence.

The cases were encountered at ages between 50 and 65 years. The average age was 59.2 years. The very beginning of the sixth decade opened mostly the heaviest incidence of death. This reflects that life expectancy in obesity is unfavourable.

3. Sex Incidence.

Sex incidence was equally divided between males and females.

4. Body Constitution.

Among the females there were two well-nourished and one obese. All the males were obese.

5. Race.

There were four Egyptians, one Italian and one Sudaneze.

6. Social Conditions.

All men were of the working class. Among the females, one was a house-wife, one a widow and one of uncertain character.

7. Habits.

A history of alcohol was obtained in five out of six cases (83.3%), and 50% of the patients were habitual drunkards.

8. Family History of Obesity.

A family history of obesity was obtained in half the cases.

9. Associated Diseases.

In all the cases, no other diseases were encountered, either to cause death per se, or to share in the cause of death. The only recognisable pathological finding in all cases was adiposity of the heart.

No history of diabetes was obtained in any case.

10. Symptoms, Time, State and Mode of Death.

(A) Symptoms.

In the four cases which were witnessed at time of death, severe precordial pain and dyspnoea were the presenting symptoms. Duration of symptoms in two cases with myocardial rupture was from ten to thirty minutes. Duration of symptoms in two cases without

myocardial /

myocardial rupture was from forty-five to ninety minutes.

(B) Time of Death.

Two cases were found dead in bed. Two persons died while at work. One case developed symptoms while on a motor car journey. One case died after a late supper.

(C) State and Mode of Death.

In two cases, death apparently was symptomless and occurred at rest, as the persons were found dead in bed, although in one case (female Sudaneze), considerable bodily exertion had previously taken place.

Exertion, such as may be caused by a motor car journey, is likely to be a precipitating factor to acute heart failure in these cases, especially in fairly old persons.

One case died under ordinary conditions, no physical exertion being implicated.

In the two cases in which myocardial rupture occurred, severe physical exertion preceded the rupture.

11. Pathological Findings:

In all cases a severe degree of adiposity of the heart was encountered. The right ventricle was mainly infiltrated by fat in all cases. The lateral wall of the left ventricle and the interventricular septum were involved to a slight degree on two occasions and one occasion respectively.

The hearts weighed from 280 to 420 gm. the average weight being 360 gm.

The coronary arteries presented a slight degree of atheroma, and their lumina were patent throughout.

No valvular lesions were encountered.

The other organs of the body presented no lesions to account for death of the patients, and in absence of such, the cardiac condition was justifiably considered the sole cause of death.

It is interesting to note that in none of the cases was fat embolism found in the lungs.

12. Cause of Death.

In four cases the cause of death was acute myocardial failure.

In two cases death occurred from cardiac tamponade due to haemopericardium resulting from myocardial rupture.

C O M P A R A T I V E S T U D Y

The condition of adiposity of the heart is very seldom diagnosed during life. Its clinical manifestations are usually hidden in those of other associated diseases, and even when present by themselves are frequently designated to some other disorder pertaining to the heart. In so much as its symptomatology is obscure, its clinical recognition is frequently overlooked /

overlooked. Nevertheless it is a recognised pathological entity.

AETIOLOGY:

1. Obesity.

It is believed by all authors that obesity is the determining cause of the condition.

(A) Pathological Definition of Obesity.

Smith and Willius stated of obesity, "is a term applied to a general condition in which there is an abundant accumulation of fat in the normal regions for storage or the fat depots of the body."

They gave the site for fat storage in the sub-cutaneous connective tissues, the omentum, the mesentery, the bone marrow, the extraperitoneal tissues, the perinephric tissues, the tissues about the orbits and to a lesser extent the connective tissue beneath the epicardium. According to them, fat is commonly deposited over the base of the heart, over the auriculo-ventricular groove, around the bases of the great vessels, along the distribution of the coronary arteries, along the interventricular sulci, and over the right ventricle, especially along its right border and anterior surface and at the apex.

(B) Causes of Obesity.

Obesity is generally due to excessive ingestion of fats and to a lesser degree of carbohydrates. The interference with the proper utilisation of fat

is apparently greatly responsible for the accumulation of adipose tissue.

It is not intended to discuss here these processes in any detail, the discussion being mainly restricted to adiposity of the heart. However, it is stated by many authors, in connection with adiposity in general, that lack of exercise and advancing years are among the aetiological factors predisposing to obesity. Lack of exercise was not a factor predisposing to obesity in at least half the cases in this series. Two men were actually engaged in heavy manual work, and the third man (cabman) was certainly a person with activities. The ages of the individuals, although most of them were in the sixth decade, are not the ages to be designated as advanced. Therefore, it is suggested that, although lack of exercise and advance of age may predispose to obesity, the latter may occur even in individuals who are not idle and are engaged in work demanding a good deal of physical activity.

Endocrine dysfunction, especially of the pituitary gland may also result in obesity.

(C) Age and Obesity:

In a series of 136 cases, Smith and Willius (1933) stated that the average age was 52.1 years. Only four patients attained the age of 70 years or more. This is in accord with the present series.

(D) /

(D) Sex and Obesity.

The female sex is declared more prone to develop adiposity than the male sex. Smith and Willius gave a ratio of approximately 2 : 1. The present series is not in accord with this statement.

(E) Race and Obesity.

Of race, Hebrews are qualified of possessing a tendency to obesity. No explanation was offered in this connection.

In the present series, although all the patients were obese, no such racial factor was disclosed.

(F) Constitution and Predisposition to Obesity.

It is an every day observation that some people eat a diet of a high caloric value and yet do not put on fat. This suggests a constitutional factor of the make-up of the body of certain individuals. Such a factor undoubtedly predisposes to obesity. Its nature, although not disclosed, is suggested to be of a biological aspect.

(G) Heredity and Obesity.

It has been noticed that obesity runs in certain families. A genetic factor seems to be the only explanation (Gurney 1936). The diet of these people may be no different from that of others, yet they are more prone to put on weight in varying degrees, and some of them become certainly obese.

It has also been noticed that certain members of

these /

these families are quite obese, while others are of a normal bodily build, although they eat and share the same kind of food. The only explanation for such an individual particularity, even in the presence of a hereditary factor, is the possession of an individual constitutional factor predisposing to obesity.

To sum up, the basic opinion of most authors is that obesity is the result of an accumulation of fat due to intake of a greater amount of food than is expended in the production of energy, irrespective of other aetiological factors, which may or may not be of significance in some cases.

2. Diabetes Mellitus.

The association of obesity in general and diabetes mellitus is a common observation.

This link is maintained by some authors as being of significance in adiposity of the heart (Spain and Cathcart 1946).

Smith and Willius recognised an incidence of 3%. Saphir and Corrigan (1933) in a series of 58 cases, found few of their patients had given a clinical evidence of diabetes mellitus.

In this series of six cases, no clinical evidence of diabetes mellitus was disclosed. Perhaps in a larger series the condition may have been encountered.

However, it should be noted that diabetes mellitus

is /

is a disease not only associated with obesity, but also with atherosclerosis in general, and it by no means, per se, infers in particular a direct relationship to obesity of the heart.

3. Alcohol.

Chronic alcoholism, especially indulgence in drinks such as malt liquors and sweet wines, is held responsible for adiposity of the heart (Powell 1909, Spain and Cathcart 1946 and Beattie and Dixon, 1948). Saphir and Corrigan described a case of alcoholic cirrhosis of the liver combined with adiposity of the heart.

In the present series this factor was very significant.

Relation between the Weight of the Heart and Body Weight.

A definite correlation between the weight of the heart and body weight was determined by Smith (1928). The ratio for the males was found to be 0.43% and that for the females 0.4%. The weight of the heart was found to increase with increase in the weight of the body.

On this basis, Smith and Willius (1933) found in a series of cases of adiposity of the heart that the weight of the hearts in an appreciable proportion of obese persons is less than demanded by the height and weight of the body. They are of the opinion that

this /

this disproportional decrease in cardiac weight may be responsible to a considerable degree for a circulatory inadequacy of obese persons. They also found that the cardiac weight tends to parallel the increase in body weight to a certain point (about 104.5 Kg.), and when the body weight increases beyond this, the cardiac weight does not increase proportionately. However, in their series they found a certain relation between the excess of epicardial fat and the degree of general obesity.

The Heart in Obesity with Hypertension.

According to Smith and Willius, the weight of the heart in this combination was found, on the average, to show an increase of 91 gm. over the control group.

Intracellular Fat in Cardiac Muscle.

It was stated by Master (1923) that normal hearts contain a certain amount of microscopically visible fat within the cytoplasm of the cells.

Smith and Willius estimated the amount of intracellular fat in their cases of adiposity and have found that it was as high as 40% in the muscle of the right ventricle. In analysing the fat of the left ventricle it was found to be within normal limits (11.6 to 23.9%), the normal being, in a control series varying from 12% to 20%.

Pathological Observations.

Pathological Observations:

It was found by authors who have already been referred to that adiposity of the heart occurs most commonly in the wall of the right ventricle. Some described adiposity in other sites, such as the left ventricle and the interventricular septum, in conjunction with adiposity of the right ventricle.

The adipose tissue finally replaces the heart muscle.

The weight of the hearts in Saphir and Corrigan's cases varied from 200 - 800 gm., the majority weighing between 300gm and 400gm.

In this series, the average heart weight was 360 gm., the largest heart weighing 420 gm.

Mortality in Adiposity of the Heart.

Smith and Willius found a mortality of 14% in cases of adiposity of the heart in which there were no other associated conditions to precipitate death. They also emphasised the risk of pulmonary embolism in their cases (19% incidence.).

Cause and Mode of Death.

Death occurs when replacement of cardiac muscle by fat involves a large proportion of the muscle. Death usually results from acute right-sided heart failure. Rupture of the heart muscle may occur in

cases /

cases with extreme myocardial adiposity resulting in cardiac tamponade due to haemopericardium.

According to most authors, death is usually sudden.

Keane and Maxwell (1940), described a case in which the deceased, while washing, fell down and died immediately.

At autopsy the heart was found infiltrated with fat and a tear measuring $2\frac{1}{2}$ inches was seen in the left ventricle.

Friedman and White (1944), described a myocardial rupture occurring in both the right and the left ventricles in one patient. The deceased was an aged female and she died a few minutes after rupture.

Fatal Symptoms:

These are often absent. Death usually occurs without premonitory symptoms of heart failure, (Saphir and Corrigan, Brouardel, and Ribbert). On many occasions it occurs suddenly and may only be preceded by dyspnoea.

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CHAPTER IV

Historical Review

The first record of myocarditis is found in the autopsy of a patient who died of a disease which is now called the acute infectious disease of the heart. The first description of the disease was given by Virchow in 1858. He described it as a disease of the heart muscle, which is now called the myocardium. The disease is now called myocarditis.

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Virchow (1858-1859) in his description of the disease

described it as a disease of the heart muscle, which is now called the myocardium.

MYOCARDITIS

The disease is now called myocarditis.

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M Y O C A R D I T I S

Historical Review:

From a review of literature it seems that old students of medicine were not familiar with this condition as a pathological entity. However, the size of the heart and the bulk of its muscle attracted their attention at the necropsy.

Massa of Italy in 1534 quite accurately described hypertrophy of the heart. Baillou (1538-1616) referred to dilatation of the heart as "aneurysm of the heart", (Herrick 1942).

Nicholas (1593-1674) in his account of what was probably a diphtheria said, "--- the heart is not exhilarated further; much less easily it digests the soot but is seen to be spread by the strong force and to come forth, like some smoke stirred by a violent wind storm or from damp and dark hovel or from smouldering coal. Indeed the heart becomes faint with such breathing and thus the vital heart becomes faint; so that the vapour retained or the air falsely mixed either oppress or kill a man" (Major, 1945).

Senac (1693-1770), recognised that inflammation may attack the pericardium as well as the lining of the cavities and the muscular structure. (Herrick 1942).

The first case described in the literature which might possibly have been a case of syphilitic myocarditis, was reported by Lancisius, 1718 (Saphir 1932).

It was a case of an aneurysm of the heart in a patient who had received treatment with mercury.

Ricord in 1845 is credited with having been the first to describe gumma of the heart (Saphir 1932).

Wilks, as stated by Saphir, presented before the Pathological Society of London in 1856, a case referred to as the first case of gumma of the heart.

Wagner (1866), Fowler (1868), Morgan (1872), Jullian (1879), Ashby (1887), Lang (1889), and many others described myocardial lesions in relation to syphilis. Many cases of sudden death were reported by a number of them.

C A S E M A T E R I A L

This series included 32 cases accounting for 8.8% of sudden cardiac death.

The cases are divided into two groups:-

- (a) Cases occurring in children.
- (b) Cases occurring in adults.

The second group comprised eight special cases autopsied in prisons. These are grouped separately.

A description of these cases, in some detail, is illustrated in the following four tables.

MYOCARDITIS

ADULT CASES

TABLE I

DISEASES	No. of Cases	Age	Sex	Fatal Symptoms	Duration	Previous History	Occupation	Pathological Findings
Influenza	6	55	M	Sternal Pain Dyspnoea	20 minutes	Influenza 18 weeks	Blacksmith	Heart 0 L.V. + Cloud. Swell. & Fat. Deg.
		50	M	Sternal Pain Dyspnoea	3 hours	Influenza 1 year	Porter	Heart Flabby 0 L.V. + Fat. Deg.
		30	F	Collapse	1 hour	Influenza 1 year	Shop Assistant	Heart Flabby 0 Myocard. Scar.
		45	F	Sternal pain Dyspnoea	6 hours	Influenza 16 months	House- Keeper	Heart Flabby 0 L.V. + Myocard. Scar.
		30	F	Collapse	30 minutes	Influenza 2 weeks	Clerk	Heart Flabby 0 Cloudy Swelling.
		26	F	Dyspnoea Cough	4 hours	Influenza 6 weeks	Labourer	Heart Flabby 0 Cloudy Swelling.
Malaria	2	30	M	Dyspnoea	3 hours	Malaria 6 months	?	Heart Flabby 0 Fat. Deg.
		42	F	Chest pain	30 minutes	Malaria 10 weeks	?	Heart Flabby R.V. 0 Fat. Deg.

R.V. = Right Ventricle

O = Dilated

L.V. = Left Ventricle

+ = Hypertrophy

Fl. = Flabby

M Y O C A R D I T I S

ADULT CASES

T A B L E II

DISEASES	No. of Cases	Age	Sex	Fatal Symptoms	Duration	Previous History	Occupation	Pathological Findings
Typhoid Fever	3	30	M	Collapse Fall from Ladder	3 hours	Typhoid Fever 12 weeks	Salesman	Heart Flabby 0 Pale Cloud. Swell.
		21	M	Chest pain sweat	1½ hours	Typh. F. 10 weeks	Milkman	Heart Flabby 0 Pale Cloud. Swell.
		18	F	Dyspnoea	?	Typh. F. 16 weeks	Maid	Heart Flabby 0 Pale Fat. Deg.
Rheumatic Fever	2	26	F	Dyspnoea	4 hours	Rheum. F. at ages 18, 20, 24, years	House- wife	Heart Flabby 0 Myocard. Scar. Mitral Stenosis.
		19	F	Dyspnoea vomiting sweat	2 hours	Rheum. F. 6 months	maid	Heart Flabby 0 Fatty degeneration
Pulmonary tubercu- losis	2	35	M	Collapse in M.O.P.D.	Few minutes	-----	?	Healed T.B. L. & R. lung Heart 0 Myocard. Scar.
		22	F	Sternal pain Dyspnoea	1 hour	Haemoptysis 5 & 3 yrs. ago	House- wife	Healed tuberculosis Heart Flabby 0.
Relapsing Fever.	1	20	M	Collapse at work	6 hours	?	Plumber	Heart Fl. 0 C.S. & F.D. (Spir. recurrentis. spleen film.)

PRISON CASES

T A B L E III

DISEASES	No. of Cases	Age	Sex	Fatal Symptoms	Duration	Previous Hospitalisation History	Imprisonment	Work	Pathological Findings
Influenza	2	35	M	Pain L. chest Cough, nose bleeding, dyspnoea	2 hours	Once Influenza 6 weeks ago	20 months	Under Detention	Heart. Fl. O. pale brown. L.V.+ Cloudy swelling & Fatty degeneration
		40	M	Sternal pain dyspnoea	4 hours	Once influenza 3 weeks ago	18 months	Under Detention	Heart Fl. O. pale Fatty degeneration
Dysentery	2	28	M	Sternal pain Diarrhoea 2 days prev.	$\frac{1}{2}$ hour	twice Bacill. Dysent.	6 months	Hard Labour	Heart + L.V. O. Myocard. scar.
		35	M	Vomiting, Abd. pain. Dyspnoea	1 hour	twice Bacill. Dysent.	26 months	Hard Labour	Heart Fl. O. Myocard. scar. (amoebic ulcers)
Malaria	1	30	M	Dropped dead during work	?	None. 3 malaria attacks last year	3 months	Hard Labour	Heart Fl. R.V. O Fatty degeneration
Rheumatic fever	1	23	F	? Died in bath	?	None. Rh. F. aet 12	1 month	Under Detention	Heart Fl. L.V. O Scars L.V. Mit. Sten
Pulmonary tuberculosis	1	30	F	? found dead in bed	?	Twice anal fistula	16 months	Under Detention	Healed T.B. L. lung Heart+ Fl.O Myocard scar.
Unknown	1	27	M	Chest pain Dyspnoea	15 minutes	None	20 days	Under Detention	Heart Fl. O. Scars L.V. anterior wall

M Y O C A R D I T I S

CASES OCCURRING IN CHILDREN

T A B L E IV

DISEASES	No. of Cases	Age	Sex	Fatal Symptoms	Duration	Previous History	Pathological Findings
Diphtheria	5	9	M	Collapse After dinner	?	Diphtheria 12 weeks	Heart Flabby 0 pink Fatty degeneration.
		8	M	Sternal pain (at play)	10 minutes	Diphtheria 6 weeks	Heart 0 pale gray Cloud. Swell & Pet. Haem.
		12	M	Dyspnoea (at play)	20 minutes	Diphtheria 6 weeks	Heart 0 pale cloudy swelling.
		10	F	Chest pain. vomiting	1 - 2 hours	Diphtheria 15 months	Heart flabby 0 pale fatty degeneration.
		7	F	Found dead in bed	?	Diphtheria 6 months	Heart fl. 0 yellow Fatty degeneration
Rheumatic Fever	1	11	M	Chest pain dyspnoea	3 hours	Rh. fever at ages 8 & 10 years	Heart flabby 0 pale Myocard. scar No stenosis
Relapsing Fever	1	11	F	Dyspnoea Sternal pain (at play)	1½ hours	Whooping cough	Heart flabby 0 pink cloudy swelling (spir. rec. spleen film)
Measles	1	7	M	-----	-----	Measles 3 weeks	Heart flabby 0 pale cloudy swelling

DISCUSSION AND COMPARATIVE STUDY

(A) Incidence of Myocarditis.

Little is known of the frequency of myocarditis in autopsy material.

According to Saphir (1941) the condition was encountered 240 times in 5,626 autopsies on routine section of the heart at the Michael Reese Hospital, Chicago. This gives an incidence of 4.2%. He also referred to Chudějová (1933), who, in reviewing autopsy material in 8,474 cases, mentioned 221 instances of myocarditis but included in the series certain instances of arteriosclerosis, kyphosis and other conditions. However, with these added diseases it is noted that the incidence of myocarditis in Chudejová's series does not exceed 2.6%.

Albert (1938) in 113 instances of various acute infectious diseases found myocarditis in 46 cases, an incidence of 40.7%. It will be noted here that the high incidence was proportional to the acuteness of the illnesses. Brown and Hunt (1940) found acute myocarditis in 58 of 625 hearts examined microscopically; an incidence of 9.12%.

Lisa (1939) reviewed the autopsies on 41 patients who died suddenly. In 36 cases there was acute myocarditis; an incidence of 87.8%. His findings are rather different from the commonly accepted

beliefs /

beliefs of the present day in that the chief aetiological factor producing myocardial changes in these cases was infection, whereas arteriosclerosis played a minor role. His figures are interesting and worth quoting:

1. Arteriosclerotic changes..... 6
2. Infectious process directly
affecting the heart.....14
3. Toxic process, secondary to acute
pneumonia.....12
4. Myocardial changes, possibly
toxic..... 6
5. Undetermined aetiology..... 1
6. No demonstrable changes to explain
death..... 2

Of the six cases of toxic myocarditis, five were due to chronic pyogenic pulmonary infection and one to acute upper respiratory infection. Lisa stated that the one common pathological finding in 39 cases was damage to the myocardial fibres. The aetiology of myocardial damage, according to Lisa, is either an infective or a toxic process. On the other hand, Hamman (1934), in a study on sudden death hardly mentioned myocarditis.

The present series is in accord with the observations of Brown and Hunt.

(B) Recognised Causes of Myocarditis in
the Present Series.

1. Influenza.

(i) Sudden /

I. Influenza:

(i) Sudden death from acute heart failure due to influenza occurred in eight cases; an incidence of 25% among all other causes of myocarditis.

(ii) The age incidence ranged between 26 to 55 years.

(iii) The cases were equally divided between males and females.

(iv) The fatal symptoms were in the following order of frequency:- sternal pain, dyspnoea, collapse and cough. One case had in addition nose bleeding which may be of no significance. The duration of symptoms was from 20 minutes to 6 hours.

(v) The intervals between the infection and death were:- 2 weeks, 3 weeks, 6 weeks (twice), 18 weeks, 1 year (twice), and 16 months.

(vi) Pathological Findings:

In addition to flabbiness and dilatation of the heart in all cases it was found that cloudy swelling and fatty degeneration of the cardiac muscle occurred up to the eighteenth week from the time incidence of the original illness, fatty degeneration up to one year, and in older cases myocardial scarring was in evidence.

Hypertrophy of the left ventricle was associated with four cases, three of the patients were above the age of 45 and one at the age of 35 years.

The review of these cases calls back the epidemic
of /

of "grippe" in Europe in 1918.

Shmorl, according to Kirch, was the first to refer to myocarditis in this connection. He described a pure form of interstitial myocarditis with foci of round cell infiltration, with hardly any degenerative changes in the muscle fibres. Three patients out of four were stated to have died suddenly.

Weiss (1933), stated that severe transient or even permanent myocardial damage may follow influenza and other types of respiratory infections.

Werckmeister-Freund (1932), reported on a case of a 21 year old man who died suddenly. He had grippe $1\frac{1}{2}$ years before his death. At autopsy severe degeneration of the muscle fibres with foci of necrosis and interstitial oedema was found. Fibroblast proliferation and lymphocytic and polymorphnuclear cell infiltration were among the findings.

An accute diffuse inflammatory process was encountered in two cases described by Craven et al (1940).

Roulet (1935), described two instances of sudden death in which foci of degeneration and scarring were found in the myocardium.

Kornblit, in a clinical study of 1,212 patients with influenza, stated that 7% showed symptoms of cardiac involvement.

Brooks stated that myocardial degeneration occurs

frequently /

frequently in influenza, especially in the stage of convalescence, and that sudden death or chronic crippling of the heart is likely to follow this implication.

The present series is in accord with Brook's statement with the exception that none of the patients lived long to be a cardiac invalid.

On the other hand, Hamburger stated that involvement of the heart in influenza is infrequent, particularly when influenza is contrasted with rheumatic fever, syphilis or diphtheria.

Helwig and Wilhelmy (1939) described 3 cases, one of a man, 32 years of age, who died suddenly; the second was also of a male aged 43 years, who died six hours after fatal symptoms, and the third of a lad aged 13 years, who died 27 hours after operation on his foot. The second case proved to have had influenza 11 years before death. Interstitial oedema, cloudy swelling and fragmentation of the heart muscle were found in the first and third cases. The second case showed myocardial scarring.

II Diphtheria:

(i) Diphtheria accounted for five deaths; an incidence of about 15.7%.

(ii) All cases occurred in children aged from 7 to 12 years.

(iii) There were three males and two females.

(iv) /

(iv) The fatal symptoms were chest pain, dyspnoea and collapse. Vomiting occurred in one case. One case was found dead in bed, two died while at play, and one after dinner. The duration of symptoms in the three recorded cases varied between 10 minutes and 2 hours.

(v) The intervals between the infection and deaths were 6 weeks (twice), 12 weeks, 6 months and 15 months.

(v) Pathological Findings:

Flabbiness and dilatation of the heart were constant findings in all cases. Cloudy swelling of the heart muscle was recognised up to 6 weeks, while fatty degeneration was found in the twelfth week and later.

Reviewing some of the literature on diphtheritic myocarditis, it was found that an incidence of 16.5% was reported by Ball (1945). His estimation was based on an electrocardiographic study recording reversible changes from day to day suggestive of the toxic nature of the disease.

The incidence of myocarditis as quoted by East and Bain (1948) may be as high as 89%. The present series agrees with the first record.

Warthin (1924), described 16 cases of diphtheritic myocarditis occurring between the ages of 2 and 40 years. In the present series the cases encountered

were /

were between 7 and 12 years. The duration of illness in Warthin's cases varied between 2 days and 6 weeks.

Nuzum (1919) described an eosinophilic myocarditis in 7 of 29 hearts, remarking that such finding was not present in many instances of death from various other infectious diseases. He also noted that the myocardial eosinophilia bore more to the severity of the clinical symptoms. A moderate degree of cloudy swelling was found in the bundle of His.

Parenchymatous degeneration of the myocardium was described by Warthin (1924), Kirch (1927) and Hoyens and Welford (1934). Kirch was able to grow the bacillus diphtheriae from the myocardium in a large number of these cases.

An incidence of diphtheritic myocarditis was recorded by Dusso (1936) as having occurred 16 years before death. He reported a case of sudden death of a man, 27 years old. This is perhaps the longest on record. The longest interval in the present series was 15 months.

Lange (1937) found evidence of healing myocarditis in a 6 year old child whose death resulted from postdiphtheritic polyneuritis.

An early change in the form of an interstitial oedema was described by Oheim (1938), the majority of the patients being from 1 to 16 years old and only 1 was 32 years old.

Albert examined the hearts of 23 children

between /

between the ages of 6 and 11 years. One half of the cases showed varying degrees of fatty degeneration of the heart muscle, and the other half showed inflammatory cells.

Burkhardt et al (1938) described a minor degree of fatty degeneration occurring in the heart.

Fukelberger (1936) produced evidence of myocardial degeneration in guinea pigs by injecting diphtheria toxins.

The pathological changes in the form of fatty degeneration are in accord with the findings in the present series.

The cases described in the present series were all faucial diphtheria. It is of interest to note that 140 cases of cutaneous diphtheria were reported by Kay and Livingwood (1946), with an incidence of myocardial damage occurring in 5% of the cases. They

encountered extensive fragmentation and disintegration of the cardiac muscle, interstitial cellular infiltration, predominantly lymphocytic in type as well as petechial haemorrhages in the endocardium, epicardium and pericardium. They concluded that myocarditis, which follows cutaneous diphtheria, is essentially identical with that which follows faucial diphtheria, but is a less frequent complication and develops rather at a later period after the onset of infection.

Among other complications of diphtheria, a high incidence/

incidence of neuritis was reported by Burkhardt et al (1938). The neuritic lesions were also found attended with a high incidence of myocarditis.

III Rheumatic Fever:

(i) Sudden death due to rheumatic myocarditis occurred in 4 cases; an incidence of 12.5%.

(ii) The ages at which deaths occurred were 11, 19, 23 and 26 years.

(iii) Three cases occurred in females and one case in a male; an incidence of 3 : 1 respectively. It was noted that the male case was the youngest in this group.

(iv) The fatal symptoms recorded in three cases were dyspnoea, chest pains and in addition vomiting and sweating occurred in one case. The fourth case died in the bath. The duration of symptoms in the three recorded cases was 2, 3, and 4 hours.

(v) The intervals between infection and death varied between 6 months and 11 years. It is interesting to note in this connection that at the extremes of these two intervals the rheumatic fever was stated to have occurred only once. In the other two cases the rheumatic fever was recurrent; one case had two attacks at two-years interval and death occurred one year after the second attack, and the other had three attacks at ages of 18, 20 and 24 years and death occurred two years after the last attack./

attack.

(vi) Pathological Findings:

Fatty degeneration occurred in the case with a short rheumatic history (6 months) with one attack of rheumatic fever.

Myocardial scarring occurred in patients with recurrent old history of rheumatic infection.

Mitral stenosis was found in 2 cases. A previous history of rheumatic infection occurred 8 and 12 years before death.

In all cases flabbiness and dilatation of the heart were constant findings.

The review of the literature on rheumatic myocarditis revealed the same incidence occurring in this series. Saphir (1941), found among 240 cases of myocarditis 30 instances of definite rheumatic myocarditis; an incidence of 12.5%.

Disturbances of cardiac rhythm and heart sounds were described by Warthin. All his cases died from heart failure.

The characteristic nodule described by Aschoff as specific for rheumatic fever was noted once by Schmorl to have occurred in a case of scarlet fever. Fahr (1930), in an extensive study of these nodules in patients dying from scarlet fever, found that they resembled somewhat those seen in rheumatic fever, but were not all identical. These observations have for some time disputed the confusion in the literature since /

since Schmorl's report.

Saphir reviewing the field of discussion on rheumatic fever, concluded that there were three stages in the development of rheumatic lesions in the myocardium.

In the first stage there is oedema of the connective tissue. Later the muscle fibres become undifferentiated and take on a homogeneous, waxy appearance. Gross and Ehrlich (1930) and Klinge (1930) observed that the connective tissue fibrils were intact, though dissociated. A moderate infiltration of round cells, neutrophil and eosinophil leucocytes were also observed. This first stage is referred to by Klinge as "early rheumatic infiltration".

In the second stage there is proliferation and hypertrophy of the connective tissue cells which eventually fuse to form the giant cells of the typical granuloma or Aschoff nodule. These nodules are characterised by the appearance of a nucleus, which is relatively large, lobular or "budding".

The third stage is marked by scarring of these granulomata. In this connection, some observers, Rössle (1935) and Morpurgo maintained that even the very early serous inflammation may heal with the formation of a scar.

Clawson (1940) found the Aschoff nodules in 13.84% of patients who had completely healed lesions

in /

in the heart valves and in 10.44% of those who had calcifications of the aortic valve (including arteriosclerotic calcification).

The importance of this somewhat detailed description lies in the fact emphasised by Skworzoff that in acute rheumatic fever, myocardial lesions in the form of exudative changes as described in (1st stage) may occur independent of Aschoff nodules, and that the clinical picture of rheumatic myocarditis at this stage is, therefore, not the result of the presence of Aschoff bodies but is caused by exudative inflammation of the myocardium. This type of myocarditis is mostly encountered in children.

This observation and other reports of the presence of Aschoff bodies in the hearts of patients who died of diseases other than rheumatic fever were used once more as further evidence against the specificity of the Aschoff body (Siegumund; Clawson (1929); Müller, quoted by Saphir 1941). However, other observations were made from experimental studies to support the view that Aschoff bodies are the most characteristic lesion of rheumatic fever, by stating that these bodies do not have a static structure, but go through an evolutionary cycle and that a considerable factor of time is probably necessary for the development of the lesion in its most characteristic form.

Nevertheless, the association of chronic

infective/

infective arthritis and myocardial damage at autopsies once more reflected a good deal of dispute as to the significance of Aschoff bodies, as it was found by Baggenstoss and Rosenberg, that in only two instances typical Aschoff bodies were seen in addition to the exudative reaction.

Sydenham's Chorea:

On the whole five cases were encountered in the literature by Fränkel, Huzelle (14 year old boy), Schroeder (21 year old woman), Stevenson, Thalhimer and Rothschild (12 year old girl) as quoted by Saphir.

In all cases typical Aschoff bodies were found in the myocardium.

IV. Pulmonary Tuberculosis:

(i) Death occurred in 3 cases, an incidence of 9.4%.

(ii) The ages encountered were 22, 30, and 35 years.

(iii) There were 2 females and 1 male.

(iv) The fatal symptoms were sudden collapse and death in a few minutes in one case, and sternal pain and dyspnoea for 1 hour in another case. The third case was found dead in bed.

(v) A history of haemoptysis 5 and 3 years previous to death was available in one case. No history was available in the other two patients with the exception of one of them who was hospitalised twice for an anal fistula, a history very suggestive of a

tuberculous /

tuberculous nature.

(vi) Pathological Findings:

Myocardial scarring was present in two cases.

In all cases the heart was flabby and dilated and an old healed tuberculous lesion was found in the lungs.

Search in the literature revealed a number of cases of tuberculous myocarditis.

According to Miller (1933), the condition was named by Boikan in 1931 "myocarditis perniciosa" on account of its fatal character.

^eLibbermeister in 1909 described 7 cases of tuberculosis with myocardial lesions.

Gaillavardin and Gravier (1928-29) stated that diagnosis in some cases is based upon fibrosis of the heart apex and chronic peritonitis.

Henry Vaquez (1924) called the disease "primary subacute". He stated that it occurs in young subjects without any history of rheumatic fever, typhoid fever or other infective disease. Dyspnoea, cyanosis, liver enlargement, gallop rhythm, dropsy, and albuminuria are associated findings. At autopsy the heart is found hypertrophied and dilated.

Miller, however, is of the opinion that some cases are syphilitic, the rest being due to tuberculosis.

V. Typhoid Fever:

(i) /

V. Typhoid Fever:

- (i) Death occurred in 3 cases; an incidence of 9.4%.
- (ii) The ages were 18, 21, and 30 years.
- (iii) There were 2 males and 1 female.
- (iv) The fatal symptoms were dyspnoea and chest pain ranging from $1\frac{1}{2}$ to 3 hours.
- (v) The illness occurred 10, 12 and 16 weeks previous to death.

(vi) Pathological Findings:

The pathological findings were cloudy swelling of the heart up to the twelfth week after illness and fatty degeneration of the cardiac muscle at the 16th week.

There is very little material provided in the literature.

Mönckeberg described lesions similar to those of diphtheritic myocarditis. Other observers such as Le Sage (1932), Albert, Cornil et al, described fatty degeneration of the cardiac muscle. Albert recognised these lesions to be followed by an inflammatory exudation in about the third week.

In evidence of these myocardial lesions, Cornil, Poursines and Giraud-Costa, constantly produced similar lesions in guinea pigs infected with the typhoid bacillus.

VI. Malaria:

(i) /

VI. Malaria:

- (i) Death occurred in 3 cases; an incidence of 9.4%.
- (ii) The ages were 30, 30, and 42 years.
- (iii) There were two males and one female.
- (iv) The fatal symptoms were dyspnoea and chest pains in two cases of $1\frac{1}{2}$ to 3 hours duration. In the third case the patient dropped dead.

(v) The illness occurred 10 weeks, 6 months and 1 year previous to death.

(vi)- Pathological Findings:

The pathological findings in all cases were fatty degeneration of the cardiac muscle.

The references in literature described fatty degeneration of the cardiac muscle in association with malaria (Dudgeon and Clark 1917, and Caskell and Miller 1919-1920). The latter authors even found the malarial parasites in cardiac muscle cells, and stated that in these cases the degenerative changes were most marked in the left ventricle.

On the other hand, Sprague (1946), stated that in his experience with several thousand cases of malaria occurring in members of the armed forces of the United States, neither acute cardiac death nor chronic cardiac disease has resulted. Nevertheless, he referred to evidence in the literature that prolonged relapsing malaria in natives of malarial

countries /

countries may result in chronic cardiac disturbances which may progress even to the point of cardiac dilatation, ventricular aneurysm and cardiac rupture. He also stated that malaria, especially of the malignant, tertian type may rarely be fatal by its direct myocardial effect, which is either toxic or anoxic through capillary coronary occlusion blocking the coronary radicles with parasites and pigment, resulting either in toxic myocarditis or fatty degeneration with especial involvement of the conductive system.

In respect of the present series, these three reported cases occurred during World War II at a time when there was a heavy outbreak of malaria (Gambia) in Upper Egypt. At that time hundreds of patients among the poor class died daily and whole families were known to have been completely wiped out overnight. No autopsies were performed on these cases. However, a few sporadic cases managed to escape to the capital towns and even to Cairo. These three cases probably represent a small proportion of deaths from this cause among the sporadic cases; a statement which is not in accord with Sprague's view.

VII. Relapsing Fever:

(i) Two cases were encountered; an incidence of 6.2%.

(ii) The ages were 11 and 20 years.

(iii) /

(iii) One case occurred in a male, the other in a female.

(iv) The fatal symptoms in the younger patient were sternal pain and dyspnoea at play, lasting six hours. The older patient collapsed at work.

(v) There was no clinical evidence of the disease in any of these two cases.

(vi) Pathological Findings:

The pathological findings were cloudy swelling and fatty degeneration of the heart muscle in addition to cardiac dilatation.

The spleen film in both cases presented a fair number of spirochaetae recurrentis.

It should be noted here that at the time of the autopsy of these two cases (1943-46) there was an outbreak of relapsing fever in Cairo and Alexandria, and many bodies were autopsied. The pathological findings in all the hearts examined were also of the same nature as described in these two cases.

Although death occurred suddenly in some of these cases, they were not included in the present series as the disease process had already been present.

VIII. Dysentery:

(i) Death occurred in two cases; an incidence of 6.2%.

(ii) The ages were 28 and 35 years.

(iii) /

(iii) Both cases were males.

(iv) The fatal symptoms were sternal and abdominal pain, dyspnoea, vomiting lasting $\frac{1}{2}$ to 1 hour.

(v) The disease occurred 6 and 26 months previous to death.

(vi) Pathological findings:

The pathological findings were cardiac dilatation, myocardial scarring. Amoebic ulcers were encountered in one case.

The literature offered few examples of this cause.

Knack reported a 20 year old soldier presenting signs of myocarditis a few weeks after having suffered an attack of dysentery. There was no autopsy.

Sidorov reported on a patient with colitis who died with symptoms of heart failure. At necropsy the heart presented fibrosis and yellow foci beneath the subepicardium. B. coli were found in the small arteries as well as in the myocardium, (Saphir 1941).

IX. Measles:

One case was encountered in a male child, aged 7 years, who died suddenly three weeks after the onset of the disease. The heart was flabby, dilated, and showed cloudy swelling. The incidence of death was 3.1%.

The literature in this connection is very scarce. True myocarditis as stated by Saphir is rare in measles. In 100 fatal cases of measles,

Dagen /

Dagen (1937), found cardiac dilatation in 24 cases. Cellular infiltration of the myocardium, chiefly of a lymphocytic type was found in 4.4% of the cases. Lowenthal recorded some cellular infiltration beneath the epicardium, similar to that found in diphtheria and scarlet fever but only in a lesser degree.

X. Myocarditis of Undetermined Aetiology:

Only one case can be given under this heading.

It occurred in a male, aged 27 years of age. His fatal symptoms were chest pain and dyspnoea lasting for 15 minutes.

At autopsy the heart was dilated and flabby. Evidence of scarring was found on the anterior wall of the left ventricle.

This single case may, perhaps, be classed under a recognised type of myocarditis, the so-called "isolated myocarditis".

A search through the literature since 1929 revealed reports of at least 10 cases, as well as a case described by Simowand Wolpaw (1935), of a male aged 23 years who died within 20 hours of admission to hospital. They concluded that in a young person with a history of rapid and progressive myocardial failure the exclusion of the ordinary aetiological factors, especially rheumatic fever, should always lead to the consideration of acute isolated myocarditis as the clinical diagnosis.

Helwig and Wilhelmy described 3 cases dying suddenly and stated that isolated myocarditis constituted a definite and not an infrequent cause of sudden death.

(C). Other Types of Myocarditis:

Pneumonia, mumps, whooping cough, syphilis and scarlet fever are among other aetiological factors.

None of these causes was encountered in this series.

In this connection, it should be noted that scarlet fever has not been encountered - to my knowledge - among Egyptians, and that during a few months service in the General Fever Hospital in Cairo, no case of scarlet fever was diagnosed.

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CHAPTER V

VALVULAR DISEASES

VALVULAR DISEASES

THE AORTIC VALVE

Historical Review:

Theophile Bonet, in his *Sepulchretum, Sive Anatomica Pratica* (1679), included a description of a case of sudden death in which the autopsy revealed an ossified aortic valve. This case, as far as can be traced, is the first description of sudden death associated with calcification of the aortic valve (Segall 1945). Bonet had not seen the case during life, nor was he present at the autopsy. He had received a piece of the aortic valve as a gift. The case was that of a Parisian tailor who fell to the ground on the street and died. At autopsy, "no disease was found anywhere except that the three semi-lunar cusps situated at the origin of the aorta from the left ventricle, were discovered to be bony".

Morgagni (1682-1771), described lesions of the aortic valve and gave clinical accounts of the cases. In one case he stated, "The valves of the aorta were indurated, and one of them even bony. The trunk of the artery itself shew'd, up and down in its internal surface, either something bony or something verging to the nature of a bone;----- It certainly cannot be denied that the aorta, in the state I have describ'd it, must resist the blood as it is driven by the heart, and, /

and, for that reason, be able to create a palpitation, a difficulty of breathing, and that sense of streightness with which the woman was tormented". (Major 1945).

Joh Wall of Worcester, in his letter to Heberden, which appeared in the Medical Transactions of the College of Physicians in 1785, described aortic valve calcification with stenosis and insufficiency. The postmortem report was dated May 30th, 1772. It contained the following as regards the lesion of the aortic valve. "Upon examining the heart, no part appeared diseased, till we opened the left ventricle; and there, the semilunar valves, placed at the origin of the aorta, were found to be perfectly ossified. They did not, as usual, lie flat upon the divided orifice of the vessel; but stood erect, and appeared to be immovable. They were entirely ossious through their whole substance; but the ossification was formed unevenly, and as it were in spines, some parts being near a line in thickness, and others thin, like a connecting membrane, but perfectly bony". (Major 1945).

Hope (1801-1841), and Stokes (1804-1878) were among others who had described stenotic lesions of the aortic valve (Major 1945).

Insufficiency of the aortic valve was described by many. The London surgeon, William Cooper (1666-1709) was the first to describe cases of aortic insufficiency in a paper entitled, "Ossification or Petrifications in the Coats of the Arteries,

particularly /

particularly in the Valves of the Great Artery", contributed to the Philosophical Transactions of the Royal Society (1706). He describes both the pathological lesions and symptoms in three cases.

"Extraordinary shortness of breath and pain about the heart" are mentioned in the case of a man who was about forty years of age (Major 1945).

Other contributors were Morgagni, Quincke, Vieussens, Corrigan and Flint. The works of the last two are quite familiar to medical men.

C A S E M A T E R I A L

Three groups of aortic valvular disease were recognised causes of sudden death:-

1. Syphilitic Aortic Disease with Aortic Incompetence.
2. Rheumatic Aortic Disease with Aortic Incompetence.
3. Calcareous Aortic Disease with Aortic Stenosis.

In this connection, it must be stated that valvular diseases encountered in all cases of natural deaths (sudden and otherwise) were 64 cases; an incidence of 3.6%.

Sudden deaths from valvular diseases occurred in 17 cases; an incidence of 26.5%. All lesions occurred in the aortic valve.

SYPHILITIC /

SYPHILITIC AORTIC DISEASE WITH
AORTIC INSUFFICIENCY

C A S E M A T E R I A L

There were 10 cases encountered in this group in which death occurred suddenly.

In none of the cases was there an aortic stenosis or organic mitral disease.

1. Incidence of Syphilitic Aortic Insufficiency among Chronic Valvular Diseases.

There were 16 other cases of syphilitic aortic insufficiency encountered. However, death in these cases was not sudden. These raise the total number to 26 cases; an incidence of 40.6%.

2. Incidence of Sudden Death in Syphilitic Aortic Insufficiency.

Out of 26 cases death was sudden on 10 occasions; an incidence of 38.5%.

3. Incidence of Sudden Death in Syphilitic Aortic Insufficiency among Sudden Deaths due to Aortic Diseases.

This was found to be 58.8%.

4. Age Incidence.

The age incidence ranged between 40 and 47 years, an average being 44.8 years.

5. Sex Incidence.

There were 8 males and 2 females; a ratio of 4 : 1 respectively.

6. Body Constitution.

There /

6. Body Constitution.

There were 6 patients of a normal build, 3 under weight and 1 well-nourished.

7. Past History.

A history of a syphilitic infection, 17, 19 and 20 years previous to death, was obtained in 3 cases. These were all males aged 40, 42 and 40 years respectively. The patients had received anti-syphilitic treatment (arsenical preparations) some years previously. No such histories were available in the rest of the cases.

8. Associated Diseases.

Two patients were chronic bronchitics, one asthmatic and one was insane, a female.

9. Fatal Symptoms, Mode of Death and Duration.

Death occurred with dramatic suddenness in two cases.

In one case the deceased, a mayor of a small town, was lately under treatment with arsenical preparations (N.A.B.). He had received, in all, three injections during the previous three weeks prior to his death. The patient was in the doctor's consulting room ready to get his fourth injection when he suddenly collapsed and dropped dead. In this particular case some of the viscerae were subjected to chemical analysis. Very minute quantities of arsenic were recovered from the liver as well as from the intestine and urine.

In /

In the second case the deceased was playing billiards and had left the table for a drink of water. Ten minutes later his body was found lying on the floor near to a drinking tap. As there was an electric wire net near to the drinking tap it was thought that he had touched the wire and had been electrocuted. At autopsy the only lesion found to account for his death was a marked degree of syphilitic aortic insufficiency.

In three cases there was severe dyspnoea following exertion. The dyspnoea continued from 30 to 90 minutes and was ultimately followed by collapse and death in about two hours from its onset.

In another three cases dyspnoea was not related to any form of exertion. It occurred during ordinary activities and continued for 15 to 90 minutes in two cases (females) and at least over two hours in the third case. Collapse preceded death in these cases. In two cases, it was ascertained that death occurred within two hours after the onset of dyspnoea.

Palpitation and dyspnoea occurred in one case. The deceased, a clerk, was seized with the attack at work and died within 30 minutes.

In one case precordial pain, giddiness and dyspnoea preceded death and were continued for over one hour. The deceased, a mason, was seized with

his /

his attack at work.

10. Pathological Findings.

In 7 cases the heart was of a globular shape. Hypertrophy of the heart occurred in all cases, ranging between 370gm. and 800gm., the average weight being 630gm.

The left ventricle was dilated in all cases. Some dilatation of the right ventricle occurred in 2 cases.

The wall of the left ventricle varied between 18mm. and 22mm. in thickness and that of the right ventricle was from 8mm. to 10mm. in thickness.

The wall of the aorta, in its proximal 4 to 5cm., was thick. The intima was wrinkled and presented puckered, pearly-coloured patches, measuring from 2cm. to 4cm. across. Atheromatous plaques were present in a moderate degree in one half of the cases, and in a severe degree in the other half. The aortic ring was dilated in all cases.

The aortic cusps were deformed. The free margins of the cusps were thickened in all cases, and everted and retracted in three cases. The constant findings were widening of the commissures, and an extension of the syphilitic process between them.

Sclerosis of the coronary ostia was noticed in two cases, both occurring in the left artery. The course of the coronary arteries was patent throughout, although /

although atheromatous plaques were present in a moderate degree.

The other valves were normal.

Microscopically there was medial degeneration. The adventitial vessels were thickened and obliterated. Infiltrations of lymphocytes and plasma cells were found around these vessels.

A moderate degree of myocardial scarring superimposed on coronary sclerosis was found in two cases. No evidence of syphilitic infiltration of the coronary arteries was found.

DISCUSSION AND COMPARATIVE STUDY

Syphilis is primarily and predominately a disease of the aorta, and the ensuing changes in the heart, with a few exceptions, are secondary to those in the aorta. The lymphatics accompanying the vasa vasorum are the most likely channels of infection. The reaction of tissues to syphilis is one of reparative fibrosis resulting in scarring and deformity.

I. Biological Development of Syphilis of the Aorta and Heart.

According to Martland (1931), there is an early invasion of the blood stream with spirochaetae pallidum before the initial lesion appears. The visible chancre appears as a local defence reaction

at /

at the original site of infection. (Martland 1931). The organisms multiply in the initial lesion in enormous numbers and are then carried either directly into the blood stream at the portal of entry or by way of the lymphatics to the regional lymph glands, and then to the thoracic duct when they are poured into the venous blood stream, reaching the right side of the heart. According to Martland, adenitis of the regional lymph nodes, such as the inguinal lymph glands, is a defensive mechanism where many organisms are destroyed by the phagocytic reticulo-endothelial cells lining these glands.

Later, when the secondary rash appears, there is, so to speak, a stage of septicaemia, or spirochaetaemia, (Maher 1931 and Martland 1931), when the organisms in large numbers invade every tissue of the body accessible to the blood stream. This stage does not last long. As it passes away the various organs attempt to rid themselves of the organisms by way of filtration through the phagocytic properties of reticulo-endothelial cells. The spleen and lungs in that respect are very successful. The latter are drained by way of their lymphatics to the peribronchial and regional nodes of the mediastinum.

As lymphatic drainage from the heart is scanty, the organisms localise in this situation. Warthin's observations are in accord with an active specific infiltration of the heart muscle at this stage.

According /

According to McLester (quoted by Martland), the heart at this stage is easily excited and quickly exhausted, tachycardia, bradycardia, arrhythmias and syncope being manifestations of such a disorder in about two-thirds of the patients. Martland, Brooks (1925) and Wile (1930) hold that such lesions of the heart are rare in early syphilis.

II. Review of some Pathological Points of Importance in Syphilitic Affections of the Aorta and Coronary Arteries.

The general view held by most authors is that the vasa vasorum in the adventitia is frequently involved first. The lesion is in the form of an obliterative arteritis with perivascular infiltration of lymphocytes and plasma cells and consequent scarring. Involvement of the media at this stage is usually minimal (Saphir and Scott 1931). The medial changes appear to be secondary and attributable to nutritional disturbances.

MacMeans (1931), on the other hand, holds that in syphilitic aortitis the intima is involved primarily from the blood stream, although it may be also involved from the adventitia through the vasa vasorum. Nevertheless, he admits that histologically the lesions are the same.

In argument, the frequency with which syphilis attacks the root of the aorta on account of the rich supply of the vasa vasorum in this region, is taken

in /

in evidence of an early involvement of the adventitia by the syphilitic process (Saphir and Scott).

Of the perivascular infiltration and scarring, Carr (1931) is of the opinion that it is very difficult to differentiate these lesions in syphilitic aortitis from those in rheumatic aortitis. However, he states that in the rheumatic type the lesions are more strictly confined to the adventitia and probably do not invade the media beyond its outer third. He, therefore, concludes that the diagnosis rests largely upon the history and stigmata of syphilis in one group and the history and the presence of other signs of rheumatic cardiac disease in the other.

Maher (1931) found that the coronary arteries and their branches may also be involved by the syphilitic process in the aorta, and that the extent of invasion may even reach the subintima of these arteries. He also found that the infiltration involves the first few centimeters of the course of the arteries, and as the arteries subdivide, the infiltration becomes less frequent and smaller in size.

In the present series, the coronaries were found free of any syphilitic infiltrations.

III. Observations on the Aortic Ring in Aortic Insufficiency.

The general opinion is that regurgitation in syphilitic heart disease is in part due to deformity

in the valve and in part due to dilatation of the aortic ring, and that these two elements are very definitely due to syphilis.

Reid (1931) explains the insufficiency of the valve as due to yielding of the muscular ring round the aortic orifice.

Mallory (1932) on the other hand, in checking over all the cases of involvement of the aortic valve in syphilitic aortitis, found that in three-quarters of the cases the valve ring was normal in diameter, and in the remaining quarter of the cases in which the valve circumference was above normal all other valves in the heart were equally above normal. He, therefore, expresses the opinion that dilatation of the ring depends upon generalised cardiac dilatation and not upon the direct effect of the luetic process. He also, states that syphilitic aortitis, per se, does not cause aortic regurgitation unless there is a deformity of the valve, and that dilatation of the aorta above the valve does not interfere with the function of the valve.

Martland (1931) does not believe that the production of regurgitation by dilatation of the aortic orifice is of much significance. He holds that syphilitic infiltration between the commissures of the aortic cusp is the determining factor in the production of regurgitation.

In this connection, dilatation of the aortic ring was noted in all cases in the present series. Widening of the commissures was also a constant finding. The left ventricle was dilatated in all cases, although no abnormalities of the other valves were found.

IV. Cardiac Hypertrophy in Aortic Insufficiency.

The main myocardial lesion in aortic insufficiency is cardiac hypertrophy. In the present series, this was found in all cases. Fibrosis of the cardiac muscle due to superimposed coronary sclerosis was found only twice.

In agreement with the above statement, Clawson and Bell (1927), in a study of 28 cases of syphilitic aortic insufficiency stated that, "With the exception of the hypertrophy, we found no anatomic change in the myocardium which seems sufficient to cause death".

Martland (1931), in a series of 36 cases found cardiac hypertrophy to be the outstanding myocardial change in all cases. He also described some degree of myocardial scarring due to superimposed arteriosclerosis.

Carr (1931), in a series of 119 autopsies found evidence of cardiac hypertrophy in 95 of the cases. Myocardial scarring was found in six cases only.

The following table illustrates cardiac weights recorded by three authors in contrast to the weights recorded /

recorded in the present series.

Cardiac Weights

AUTHOR	Smallest gm.	Largest gm.	Average gm.
Martland (1931)	360	1160	600
Carr (1931)	300	1060	
Saphir and Scott (1931)	400	1050	600
Present series	320	820	630

V. Incidence of Aortic Insufficiency among Syphilitic Diseases of the Aorta.

VI. Age Incidence.

VII. Sex Incidence.

According to six authors, these are expressed in the following table:-

AUTHOR	Incidence Per cent	Age years	Sex
			M : F
Saphir and Scott (1931)	24.3		
Martland (1931)	33.7	40-50	5 : 1
Carr (1931)	37.2		
Willius (1931)	59.0	49	
Scott (1931)	60.0	40-50	6 : 1
Reid (1931)		47.9	5.5: 1

The present series is in accord with the above findings.

However, exceptions to the general rule may occasionally occur. Aortic insufficiency was

reported /

reported by Scott in a negro girl aged 17 years.
Willius reported on a case, aged 16 years.

VIII. Race:

Most authors agree that syphilitic infection is commoner among the negroes than among the whites. This is probably due to early puberty in the black races as well as to lack of adequate anti-syphilitic treatment among them.

Martland (1931) found a ratio of 1.25 : 1 respectively.

Turner (quoted by Maynard et al 1935) found a ratio of 1.6 : 1.

Maynard, on the other hand, recorded a greater incidence of syphilitic affections among the whites than among the negroes. He found an incidence of 4.3 : 1 respectively, but he had noted that his records covered a community where the number of the negroes is small.

This variation, therefore, does not contradict the general rule.

A racial factor was not disclosed in the present series.

IX. Fatal Symptoms in Aortic Insufficiency.

According to Scott (1931), in a series of 107 patients, the symptoms complained of by the vast majority of patients were those of the onset of heart failure; dyspnoea on exertion and palpitation. Precordial

and /

and substernal pain or anginal attacks were seldom noticed except in those cases of marked narrowing of the coronary arteries, and even in the latter cases, chest pain was not always present. This is consistent with the findings in the present series.

Reid (1931) stated that symptoms of the disease may be absent in about one quarter of the patients in whom the disease was disclosed at necropsy. This remark is in accord with the findings in this series; two patients (one fifth of the cases) died suddenly without complaining of any symptoms.

In Reid's series, chest pain formed 28% of the symptoms, and pains of anginal type 4% only. Dyspnoea was a prominent symptom. Levine (1921) attributed 7% of anginal pains to syphilis.

In the present series, precordial pain occurred in one case, accounting for 10% of the symptoms.

Willius reported painful cardiac seizures in 25% of the cases. This is probably the highest ratio reported.

X. Causes of Sudden Death in Aortic Insufficiency.

Sudden death in these cases may occur in a fairly large proportion of cases.

Martland is of the opinion that it is either due to acute cardiac dilatation or cardiac failure of an anginoid type, and that the latter is especially liable

to /

to occur when there are associated lesions about the orifices of the coronaries, or a superimposed coronary arteriosclerosis.

According to Leary (1940), aortic insufficiency may be responsible for sudden death because of its effect on the cardiac blood supply, as the main coronary circulation occurs during diastole. The muscular branches of the coronary arteries are compressed during systole by the contraction of the ventricular muscle. In aortic insufficiency the closure of the aortic cusps is inadequate, with the result that there is leakage back into the left ventricle and, thus, the coronary circulation is not adequately maintained, and sudden death may be the outcome in the early stages of the disease.

An incidence of 13.6% has been reported by Willius (1931)

Martland reported an incidence of 36%.

XI. Interval between Primary Infection and Death.

The interval usually reported in the literature is 20 years.

Scott (1931) gives an average duration of 26 years. However, the shortest interval in his series was 5 years and the longest 48 years.

Maynard et al (1931) give a duration of 16 to 20 years.

Willius gives an average duration of 23 years.

In /

In the present series the average duration ascertained in 3 cases was 22 years. This is in accord with the previous findings.

XII. Duration of Life after Development of Primary Symptoms.

This could not be ascertained in the present series as no reliable history was obtained.

Martland gives an average duration of life of 6 to 12 months, and two years as a maximum. This certainly indicates a grave prognosis in these cases. However, he also states that exceptions to the rule may occur and a few patients may live many years.

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RHEUMATIC AORTIC DISEASEWITHAORTIC INCOMPETENCECase Material:

There were three cases encountered in this group.

1. Incidence among Aortic Valvular Diseases Causing Sudden Death:

Rheumatic aortic incompetence accounted for 17.7% of sudden death among aortic valvular diseases causing sudden death.

2. Age Incidence:

The ages encountered were 50, 55, and 55 years.

3. Sex Incidence:

There were two males and one female; a ratio of 2 : 1 respectively.

4. Race:

The males were Egyptians and the female was a Greek.

5. Body Constitution:

The males were muscular, the female was well nourished.

6. Occupation:

The younger male was a blacksmith, the older a farmer. The female was a housewife.

7. Past History:

A vague history of what might have been a rheumatic infection was obtained in the male cases

at /

at ages of 8 and 10 respectively. The female case was a chronic bronchitic. A history of rheumatic infection occurring at the ages of 10 and fourteen years was available.

8. Family History:

No relevant history of rheumatic infection was available in the male cases.

The female patient had a younger sister, who died at the age of 11 years with rheumatic fever.

9. Fatal Symptoms:

In the case of the blacksmith, it was stated that while at work he was seized with a severe attack of palpitation and dyspnoea and had to go home. His attack did not ease with rest and even grew more severe. A doctor who examined him one hour later, gave him an injection (?), and ordered him cachets containing luminal and papavarine hydrochloride. The deceased died about six hours after his fatal symptoms, and was stated to have coughed some blood twice before his death. The patient was stated also to have been breathless for the last nine months.

The second patient was stated to have gone swimming with some friends. About fifteen minutes later he was noticed to be in difficulty. He was immediately taken to the shore in a collapsed state, but soon expired. At autopsy no signs of drowning

were /

were found. The only lesion to account for his death was incompetence of the aortic valve.

The female patient collapsed while attending a dinner party and died thirty minutes later. It was stated that she had been quite active till the time of her death, and never had complained of any disabling symptoms.

10. Pathological Findings:

A moderate degree of ankle oedema was present in the male cases. The hearts were hypertrophied and weighed 500gm. 450gm. and 550gm. respectively; the average weight being 500gm. The left ventricle was dilated in all cases. The aortic cusps were thickened, scarred, shortened and everted. The valves were incompetent. Tiny, hard, adherent vegetations were found on the cusps of the aortic valve, situated on the ventricular surface of the cusps. Evidence of the rheumatic nature of the infection was also found.

The other valves of the heart were normal.

A marked degree of pulmonary oedema was present in the first case (blacksmith), and to a slight degree in the other two cases.

Other pathological findings, apart from a moderate degree of atheroma of the aorta were not encountered in any of the cases.

DISCUSSION AND COMPARATIVE STUDY

1. Pathogenesis of Rheumatic Fever:

(a) Precipitating Factors:

The causative organisms responsible for rheumatic fever have remained in dispute up to the present date. There is no general agreement concerning the nature of the infectious agent.

Three theories have been advanced in this sphere:-

- (i) That it is caused by the streptococcus haemolyticus.
- (ii) That it is a state of allergy.
- (iii) That it is a virus infection.

Those who hold the first view claim that haemolytic streptococcal infection precedes the development of rheumatic fever in many cases. Sheldon (1931) observed that in one epidemic of streptococcal throat infections among rheumatic children, relapses of rheumatism occurred one to three weeks after the attack. This is a true common observation, although organisms are neither demonstrated in the lesions (Aschoff nodule), nor cultivated from the blood. Similar observations were also noted by Glover (1930) and Green (1942). However, further work by Coburn and Pauli (1939) suggests that the production of specific antibodies in the blood subsequent to streptococcal throat infection is

in /

evidence of a coccal infection. The specific anti-bodies have been designated precipitins, agglutinins, and anti-haemolysins. Further support of this theory is claimed at the expense of the titre of these anti-bodies, rising steadily and persistently in rheumatic fever.

Clawson (1940) states that although the streptococcus viridans has been most commonly isolated from the blood, joints and pericardial exudates of patients with acute rheumatic fever, it is not the causative organism.

The allergy theory is again based on a streptococcal throat infection, which gives rise to a general hypersensitivity. Acute rheumatic fever is the manifestation of this state of allergy, a hypersensitivity to bacterial products (Swift et al 1928).

The pathogenesis of rheumatic fever is explained by the existence of hypersensitivity to streptococci resulting from repeated low grade infection or the persistence of foci of infection in the body. According to Scherf and Boyd, this state of allergy appears to be the most acceptable theory for the pathogenesis of rheumatic fever.

The virus theory is based on the observations of Schlesinger et al (1935). They found bodies in the pericardial exudate which resembled the elementary bodies of a virus.

Sabin (1939) isolated filterable pleuro-pneumonia - like organisms from mice. These organisms, when injected into other mice, were capable of producing arthritis in a high percentage of cases.

(b) Predisposing Factors:

Rheumatic fever is a disease of temperate climate. It is probably more common in Great Britain than anywhere else, also in North America and Germany (Sutton, 1929 and Price, 1946).

The incidence of rheumatic fever in the tropics is low (Quinn 1946). Such a low incidence of the disease is also noted in Egypt. The disease is, however, more frequent in damp towns of Lower Egypt than in Upper Egypt, and of a more frequent occurrence in towns than on the countryside.

In towns the incidence of the disease varies from one part to another depending upon the crowding of the population. No doubt overcrowding parallels poverty. Poverty, which entails inadequate food intake, hunger, malnutrition, dirt, bad clothing and exposure to cold and damp plays a large part in predisposition to the disease (Quinn, 1946, and East and Bain 1948). It was found in America that children predisposed to rheumatic fever, when given four eggs a day did not succumb to the infection, while the controls /

controls developed the disease.

II. Age Incidence:

Rheumatic fever is primarily a disease of childhood. Sutton (1929) found the peak of incidence occurring at the ages of 9 years in girls and seven years in boys.

Wilson et al (1929) recorded the peak of age incidence between 9 and 12 years.

According to White (1946) rheumatic aortic valve disease is commonest in middle age.

Clawson (1940) stated that the highest death rates occurred in the fifth decades in either completely healed or incompletely healed valves.

The present series is almost in accord with these observations. The incidence of mitral to aortic valve involvement, as stated by Clawson, is 16 : 1 in unhealed lesions and 8.8 : 1 in healed lesions respectively. The evidence from this observation suggests a longer span of life with rheumatic aortic disease than with mitral disease.

III. Sex Incidence:

Females are rated to predominate slightly over males.

Clawson (1941) gives a ratio of females to males
= 1.1 : 1

Sutton (1929) gives a ratio of females to males
= 1.3 : 1

Wilson /

Wilson et al, (1929) gives a ratio of females
to males = 1.5 : 1

White (1946) states that rheumatic aortic
valve disease is commoner in males than in
females at about a ratio of 3 : 1.

The present series is in accord with White's
statement..

IV. Race:

As regards race, Sutton (1929) stated that
certain races, such as the Chinese, Japanese, and
natives of India are seldom affected.

On the other hand, Scherf and Boyd, (1948) are
of the opinion that racial susceptibility to rheumatic
infection has not been established.

In the present series it has been noted that
the incidence of rheumatic affections in Egypt is
low.

V. Family History:

Heredity seems to play a part (Wilson and
Schweitzer (1937) and Gauld and Reid (1940)).

Wilson et al (1929) recorded a hereditary
incidence of 36%. They also referred to St. Laurence
and Faulkner and White, who reported an incidence of
24% and 8.8% respectively.

In the present series a family history of
rheumatic affection was recorded in one case.

VI. Avitaminosis:

Inadequate /

VI. Avitaminosis:

Inadequate nutrition, as well as a deficiency of vitamins (A, B, C, and even D) has been frequently asserted to favour the development of the disease (Scherf and Boyd). However, they stated that no evidence in support of avitaminosis was provided as of a significant aetiological factor predisposing to rheumatic affections. The low blood levels of vitamin C frequently reported in these cases, may be (as stated by Scherf and Boyd), due to inability to utilise the vitamin during the course of the disease.

The present series is not in accord with a vitamin deficiency theory. It is hardly expected that a person of the well-to-do class (female case) and a farmer, do not take an adequate supply of vitamins in their diet.

VII. Pathological Points of Importance:

Clawson (1940) observed that the valves of the left side of the heart were chiefly involved, (99.8%), and the aortic and mitral valves were involved with equal frequency in males.

He also noted that vegetations begin on the side of the valve where the spongiosa layer, which contains most of the blood vessels, is located, namely on the ventricular surface of the aortic and pulmonary cusps, and on the auricular surfaces of the mitral and tricuspid valves. He, therefore,

thinks /

thinks that the infection in some cases may be embolic in origin, although in other cases, is carried through the blood stream.

According to White, (1946), severe or repeated rheumatic infection, through healing by scar tissue, produces scarring, retraction and stiffening of the three borders of the aortic cusps, thereby producing regurgitation. Adhesions between the cusps at the commissures produce stenosis of the valve. A combination of regurgitation and stenosis of the valve may be encountered. A preponderent regurgitation is much more common than preponderent stenosis, in the ratio of about 5 : 1.

In accord with this statement, Scherf and Boyd state that insufficiency of the aortic valve is one of the most frequent isolated valvular lesions. They referred to Adlmuhler (1920), who reported an incidence of 19.5%. In the present series the incidence of this disorder among other cases of aortic valve diseases causing sudden death was 17.7%.

VIII. Effects of Rheumatic Aortic Incompetence on Physical Efficiency:

Patients suffering from this disorder are usually free from complaints even with marked leakage of the valve.

This is in accord with the present series.

The /

The three patients did not exhibit any disabling symptoms with the exception of the first case (blacksmith). However, this patient, in spite of a complaint of dyspnoea for the last nine months, was able to carry on his work till the time of his death.

Scherf and Boyd (1948) stated that persons with aortic insufficiency, "have but few symptoms". "They are fully active and pursue ordinary sports and may even climb mountains without symptoms. The diagnosis of an organic disease is to them a great surprise."

Jockl and Suzman (1940) described a case of a male, aged 32 years, who had combined aortic regurgitation and mitral stenosis. In spite of these lesions, he was a successful long distance runner. He had won a race of 26 miles for the British Empire games and on two occasions, entered a 54 mile race and covered the distance in good time. He also competed successfully in two races within eight days. Suzman referred to a case of a man, aged 34 years, with an aortic regurgitation of a rheumatic origin, who excels in long distance swimming.

Waifeld (1934) recorded a number of youths with aortic insufficiency, who were athletes, and even some of them were record holders.

The prognosis, therefore, in these cases is

generally /

generally good. A long duration of compensation is expected in the rheumatic type. The duration of compensation depends mainly on the condition of the myocardium and only to a slight degree upon the extent of the valvular lesion (Scharf and Boyd). Early cardiac enlargement may take place when the heart muscle is damaged by the rheumatic process, and rapid cardiac dilatation even at this stage may be injurious.

IX. Symptoms of Rheumatic Aortic Incompetence.

Symptoms, as previously stated by many authors, are usually absent.

According to Scharf and Boyd, dyspnoea may be the only complaint, and, as a rule, is of the paroxysmal, nocturnal type, typical of left ventricular failure. Exertional dyspnoea may follow very rapidly or only at a later stage with the speed with which the left ventricle fails and pulmonary stasis develops.

In the present series, dyspnoea was the only symptom in one case.

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CALCAREOUS AORTIC VALVULAR DISEASE
WITH AORTIC STENOSIS

Case Material:

There were 4 cases in this group of valvular disease all showing aortic stenosis.

A comparative study of these cases with two series by Margolis et al (1931) and Dry and Willius (1939) is self-discussing. This is shown on the following page.

It remains to state that:-

A. In the present series calcification of the aortic valves was of a marked degree, and the aorta presented a severe degree of atherosclerosis.

B. Incidence of sudden death due to calcareous aortic stenosis among sudden deaths due to aortic diseases was 23.5%.

C. State at Death:

The oldest patient was found dead on the road. The other patients died at work.

D. Occupation:

Two patients were labourers, one an engineer and one was retired.

E. Race:

All patients were Egyptians.

CALCAREOUS AORTIC VALVULAR DISEASE

Features of Disease	Margolis et al (1931)	Dry and Willius (1939)	Present Series
Incidence per cent among valv. diseases.	?	18.1	6.3
Age in Years	25 - 87	20 - 89	45 50 65 70
Sex	4.25 : 1	4.0 : 1	All males
M F			
Body Constitution	Well-nourished	?	Average Well-nourished Average Average
Rheum. Hist. Per cent		22	25
Heart weight in gms.	265 - 740	200-1,000 +	340 320 450 400
Aortic Valve Orifice	4mm.	2 - 3 mm. 6 - 7 mm.	8 mm. 10 mm. 5 mm. 5 mm.
Mitral Valve	Normal or Slight atheroma	Normal or Calcified (12%)	Normal Normal Slight atheroma. Normal
Coronary Arteries	Sclerosis	Sclerosis Occlusion once	Sclerosis
Symptoms	Dyspnoea Palpitations Precordial pains Sternal pains Weakness	Dyspnoea Palpitations Syncope Vertigo Congestive failure	Dyspnoea Palpitations Precordial pains
Sudden Death Per cent	12	17	all

DISCUSSION

Mönckeberg (1904) is credited with the original description of this disorder of the aortic valve (Dry and Willius 1939).

The disease consists of thickening and calcification of the aortic cusps producing a variable degree of distortion of the valve, and in certain instances, varying degrees of stenosis, insufficiency, or both. Usually it is associated with a marked degree of stenosis.

The process primarily involves the aortic ring, which becomes hard, roughened and irregular with calcium deposition projecting in the form of spicules on the aortic surface of the attached border of the cusp. Calcified nodules may also involve the aortic surface of the cusps near the ring rather than the free margin. The nodules may be completely covered by the endothelial lining. Fusion of the cusps is not infrequent. It was found in nearly half the cases reported by Margolis et al, involving the adjacent edges of the right and left anterior cusps. Incomplete fusion occurred in a quarter of these cases. In a few instances fusion was due to fibrous tissue. When the degree of calcification was marked it was found to have extended on the ventricular surface of the valve, but otherwise this surface remained smooth. Margolis et al also found that occasionally the process of calcification had extended from the aortic ring into the /

the immediately adjacent portions of the aorta, but had never extended into the aorta to involve the orifices of the coronary arteries. According to them stenosis occurred in about 70% of the cases. When all the three cusps were involved the valvular opening was reduced to 4mm. in diameter. Dry and Willius encountered diameters of 6 to 7 mm. and 2 to 3mm.

Calcification of the mitral cusps, as stated by Margolis et al, may also be encountered at the same time. It is usually of a slight degree and does not interfere with the function of the valve.

Atherosclerosis of the aorta, either of a slight or moderate degree, was a finding in all their cases. Fibrosis of the myocardium occurred in one third of the cases.

Types of Calcified Aortic Valve.

Two types of calcification of the aortic valve were recognised:-

1. A degenerative type purely involving the collagen fibres leading to calcification (Sohval, A.R. & Gross, L. 1936.)

This was the view advanced by Mönckeberg.

Thalhimer (1922) maintained a non-bacterial origin, considering at the same time the process of calcification as a part of that of arteriosclerosis.

Clawson /

Clawson et al (1926) in a study of fifteen cases were of the opinion that calcification of the valves was entirely unrelated to an inflammatory process; and concluded that the aetiology of this lesion was unknown. The average age incidence in their series was 54.5 years.

2. An inflammatory type of rheumatic origin.

Cabot was among those who advocated this hypothesis, although in his series of 28 cases of pure aortic disease, only three occurred in females and twenty-five in males, a different finding from that usually encountered in cases of rheumatic carditis. The age incidence in his series also did not correspond with that encountered in rheumatic fever, for only six patients of his series were less than forty years old and half the patients were above fifty years old when they were first seen.

The rheumatic aetiology is also held by Baggenstoss and Rosenberg (1941).

The number of cases in this series is too limited to justify further discussion.

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CHAPTER VI

RUPTURE OF ANEURYSM OF THE AORTA

RUPTURE OF ANEURYSM OF THE AORTA

Historical Review:

The history of aneurysm is very old and fascinating. Galen, in the second century, described false aneurysm resulting from trauma to an artery (Erichsen, 1844 and Osler, 1942). Antyllus, in the second century, devised his operation of incising and emptying such a sac enclosed between ligatures, (Osler, 1942). Fernelius, in 1542, called attention to aneurysms of internal arteries especially in the chest, or about the spleen and mesentery, when violent throbbing is seen. Aetius, in the 16th century, described lesions due to rupture of an artery without external injury, especially as seen in the neck, saying, "--- it very commonly happens to women during parturition, on account of the forcible detention of spirits", (Erichsen, 1844). Later in the same century, Ambroise Paré, suggested the relation of aneurysm to syphilis, (Major, 1932 and Boris, 1942).

Lancise, in 1728, classified aneurysms as "true" and "false" but not in the modern sense. These terms were used, in the modern sense, by Hunter in 1757, who also described the symptoms of pressure he encountered in five cases of aneurysms. He also published a detailed account of a case of aneurysmal sac pointing through the chest wall, (Boris 1942).

Donald /

Donald Monro, in 1760, in a paper on the subject of aneurysm, made the first reference in English literature to syphilis. He said, "Sometimes a scorbutic or venereal taint, or some other acrimony in the blood has been accused, (Erichsen 1844). Morgagni, in 1761, described very fully the symptoms and morbid anatomy, (Osler 1942). Corvisart, in 1812, described various signs and symptoms, and called attention to the thrill, retromanubrial dullness on percussion and inequality of the pulse.

Oliver, in 1878, described the tracheal tug, Thoma, in 1888, wrote on the pathology of aneurysm, Döhle, in 1895, described the microscopic changes in aortitis, and finally, Reuter, in 1906, describing the treponema pallidum in the wall of the aorta, is credited with establishing the importance of syphilis as a cause.

C A S E M A T E R I A L

Rupture of an aneurysm of the thoracic aorta was encountered in three cases. These are described below in the order in which they were received.

Case I.

A.El.H.H., a male, aged 45, married, fishmonger, dropped dead in a railway station

while /

while he was running along the platform to the far end of the train to secure a seat in one of the third class compartments. He had also rushed when he bought his ticket as he thought that he was late.

This evidence was given by two friends who accompanied him to the station. They stated that he was in apparent normal health previous to the time of his death and that they only noticed that he was breathless when he returned after buying his ticket. No relevant history was available from his relatives apart from a minor accident to his foot some years ago, and breathlessness on exertion for the last six months.

Autopsy on 19.11.1938: The body was that of a heavily built individual. The face was deeply cyanosed. The chest was normal in appearance.

The heart was displaced to the left and slightly downwards. The pericardial sac contained 280c.c. of dark blood, mostly fluid. A saccular aneurysm, 6c.mm. across was seen on the ascending portion of the aorta anteriorly extending to the right. A tail-shaped, recent, irregular perforation about 5mm. long was seen about the middle of the aneurysmal sac anteriorly leading into the pericardial cavity. The right pulmonary artery, the right bronchus and the superior vena cava were pressed upon. No erosion of the vertebrae was evident.

The /

The intima of the aneurysmal wall was thickly covered with recent partially clotted blood. It also showed extensive atheromatous infiltration. Adjoining parts of the intima were scarred and puckered.

The heart weighed 390 gm. and its cavities were dilated. The left ventricular wall was nearly 2cm. thick. The aortic ring was appreciably dilated. No valvular lesions were found.

The lungs were congested and oedematous. The liver was slightly enlarged and congested and the spleen was congested. The stomach was empty of food.

Case II.

A.E.L.W.A.E.L.M., a male, aged 40, married, died suddenly in presence of witnesses. He was chatting to some friends in a cafe and then for no reason ceased talking. His friends tried to arouse him but, to their astonishment he did not answer back. He was immediately carried to a first-aid hospital where he was pronounced dead.

All witnesses agreed that he was in a normal condition till the moment he ceased talking and that he was in their company for nearly two hours.

The relatives stated that he occasionally had complained of "rheumatic" pains in his back, and that the pains never disabled him, though, they were moderately severe at times. The duration of his complaint was between six months to one year. He was employed as an assistant mason.

Autopsy on 1.1.1946, revealed the following:

Deceased /

Deceased was well-developed and muscular. The face was cyanosed. The chest was of a normal appearance. The heart was displaced downwards and to the left. The pericardial sac was distended with 200c.c. of dark blood containing three recent clots each about 5cm. in diameter. A saccular aneurysm, measuring 9cm. across, presented posteriorly on the ascending portion of the aorta and extending to the right. It pressed on the adjacent structures. No erosion of the vertebrae was seen. A recent ragged rupture measuring about 3mm. in diameter was discovered in the lower portion of the aneurysmal sac anteriorly. The lining of the sac was roughened generally with atheromatous plaques. Little laminated clot was observed. Puckering and scarring of the intima of the adjacent parts of the aorta was extensive. The aortic ring was moderately dilated.

The heart weighed 360gm. The myocardium was pale brown. The ventricles were dilated, especially the left one. The wall of the left ventricle was 1.8cm. thick. The heart contained about 20c.c. of fluid blood.

No valvular lesions were present.

The lungs, liver and spleen were congested. The first were oedematous and the liver was moderately enlarged. The stomach was empty of food.

Case III

A. EL R. H., a male aged 55, single, baker, while engaged in lifting heavy trays was seized with agonising pain in the chest, had to sit down, but was found dead after a few minutes.

No previous history of such an attack of pain was given, though he always complained of an unproductive cough which was attributed to the nature of his work, being always in a hot atmosphere, and also to his weak chest. The duration of cough was three years.

Post-mortem was performed on 16.8.1946. Deceased was strongly built. The face was pale. The chest was of normal appearance.

The heart was displaced to the left. The pericardial sac contained 150c.c. of dark blood mostly clotted.

The ascending portion of the aorta presented a saccular aneurysm, 8cm. across, situated on the anterior aspect of the vessel and extending to the right. On the lower portion of the sac anteriorly, a fresh, ragged rupture, lunar in shape, 6mm. in diameter, was seen communicating between the aorta and the pericardial sac. The adjacent structure to the aneurysm was pressed on. No erosion of the vertebrae was found. The intima of the aneurysm showed old laminated clot and freshly clotted blood, as well as widespread atheromatous plaques. The adjacent /

adjacent intima presented scarring and puckering. The heart weighed 420gm. and contained about 20c.c. of fluid blood. The wall of the left ventricle was 2cm. thick. All cavities were dilated. The valves were normal. The aortic ring was greatly dilated. The lungs, liver and spleen were similar to those in case II.

Sections of the sac in the above three cases showed an extensive degeneration of the intima and media, with loss of the elastic tissue of the media. The adventitia was greatly thickened and its vessels were sclerotic, the lumena were narrow and even obliterated and an aggregation of lymphocytes and plasma cells was seen around the adventitial vessels.

A Case of An Unruptured Aneurysm of the Thoracic Aorta.

In this connection a brief account is given of a case of an unruptured aneurysm of the thoracic aorta which was found at post-mortem in a Greek male, aged 45, who died from pneumonia.

The aneurysm was of the saccular type, about 10cm. in its widest diameter, and occupied the anterior wall of the ascending portion of the thoracic aorta, extending to the right. Evidence of a syphilitic infection was found in the aneurysmal wall. Previous to his pneumonia the patient was stated to have enjoyed fairly good health except for a complaint of pain in the chest and legs of one and a half years'

duration /

duration. The patient was single and was employed as a waiter.

Aneurysms in other sites than the Thoracic Aorta.

Two more aneurysms were found at post-mortem in the abdominal aorta. Both patients were males aged 60 years. One of them had a rupture of the aneurysm and died three days after admission to hospital. The case was not diagnosed clinically. The other case was found in a patient who died from a stab wound of the chest involving the lung.

Both aneurysms were of the atherosclerotic type.

DISCUSSION

1. Incidence of Arterial Aneurysm in General

Out of 3250 cases, arterial aneurysms were found six times, an incidence of 0.18%. The incidence of thoracic aneurysm was 0.12%. All cases occurred in the aorta.

The condition occurred four times in the thoracic aorta and twice in the abdominal aorta, a ratio of 2:1. Regarding this ratio, it must be pointed out that since the total number of aneurysms encountered in the autopsies was small, it would be admittedly difficult, from a statistical point of view, to consider with much gravity that such a ratio is a

truly /

truly representative one. However, the findings apparently suggest that aneurysms of the thoracic aorta are decidedly commoner than those of the abdominal portion. To avoid further repetition in considering other data, the smallness of the number of aneurysms under discussion shall be kept in mind.

No arterial aneurysms in other sites were encountered.

2. Age Incidence.

Syphilitic aneurysms of the arch of the aorta were encountered in post-mortem at ages of 40, 45, 45, and 55 years, an average was 46.3 years of age, and a maximum incidence was at the age of 45 years.

As regards the age incidence of the occurrence of the condition, this may be drawn from an analysis of the histories in the four cases. Thus, assuming that the duration of the symptoms is consistent with the presence of an aneurysm, it will be found that the average age incidence in three cases (excluding the patient aged 40), was 46.7 years of age. However, if it is presumed that an aneurysm in its early period of existence is not likely to give rise to warning symptoms to the patient, it may, therefore, be suggested that an early "silent" phase may exist with some aneurysms. In taking such a "silent" phase /

phase into account in these cases, the average age incidence may drop to under the age of 46.7 years. Nevertheless, in case II, the age incidence was under 40 years of age.

In this connection there are other determining factors which may cause either a delay or progress of the development of the aneurysm. In this respect the question of an antisyphilitic treatment is most important, and also whether or not such a treatment was secured early and efficiently. The state of the arterial tension and the class of the patients are other determining factors.

In the cases under discussion, consideration of these points suggests that the patients were ignorant of their venereal infections, and therefore, it may be assumed that the disease process was fairly rapid and that consequently the aneurysms were formed at an earlier date than is found in these cases. In this respect an age of 40 years may be approximately correct on the average.

3. Sex Incidence.

All cases were found in males. This is decidedly a definite evidence of the predominance of the condition in the male over the female sex. Unfortunately no sex ratio is available in this series as the condition was encountered in males alone.

Why /

Why should the male sex predominate? This may be argued on the following points, though no material evidence could be brought in support:-

- (a) Syphilitic infection in the male sex may be intensive and progressive, while it is perhaps of a milder nature in the female.
- (b) The media of the arteries in the female sex is gifted with a peculiar quality, structural or otherwise, against infection.
- (c) Males, by nature, are labourious and, therefore, are more predisposed to frequent strain on their arteries, which readily yield under such a specific infection and its deleterious effect on the media.
- (d) Particular care or fear of the female sex against such a dreadful disease.
- (e) Early and adequate treatment in syphilitic females, especially in married woman who usually seek early advice on incidence of repeated abortion.
An analysis of some of these suggestions by Venereal and Maternity Departments, and also in Social Service Centres may be of value.
- (f) Hard work and strain in the male sex may partly satisfy an explanation. In this connection, one would wonder what would be the incidence of the condition in the future, say in a hundred years to come, when mechanised and better scientific devices would be expected to relieve or abolish manual work. A clue to the answer of this question may parallel a decline in the incidence of aneurysms if such has actually occurred during the last twenty-five years of advancing science.

4. Race.

Three cases occurred in Egyptians and one in a Greek. As regards race, no definite evidence could be drawn from these cases, firstly because of the

majority /

majority of the population being Egyptians and, therefore, a higher percentage of aneurysms would be expected to be found among them, and secondly because the number of cases under discussion is very small.

5. Occupation.

All four cases occurred in working class people. In two of the three cases in which ruptures occurred, employment was of a labourious nature.

6. Determining Causes.

Syphilis was responsible for all aneurysms of the thoracic aorta. The two cases of aneurysms of the abdominal aorta, which are not under study, were of an atherosclerotic nature. The incidence of all aneurysms of the aorta in respect of syphilitic and atherosclerotic affections was 2:1 respectively while the incidence of syphilis in the production of aneurysm of the thoracic aorta was 100%. So far no other determining causes were encountered. That syphilis stands out alone as the sole determining cause of thoracic arterial aneurysm is rather unique in the face of the fact that atherosclerosis, as pointed out previously, outplaced all other affections of the arteries. Therefore, aneurysm of the thoracic aorta, subsequent to atherosclerosis may be regarded as minimal.

7. Incidence in the Various Portions of the Thoracic Aorta.

The four cases encountered in the thoracic portion of the aorta were all in the arch, namely its ascending portion. As none were encountered anywhere else on the other portions of the thoracic aorta, it is suggested that the ascending portion of the arch is the most commonly involved site in aneurysms. This observation is in accordance with the fact that syphilitic aortitis first involves the ascending portion of the aorta, with the result that the media in this region suffers degenerative changes long before the distal parts, which may be involved later in the disease.

8. Relation of Aneurysm to the Wall of the Artery.

In the three cases under discussion, the aneurysms occurred twice on the anterior wall of the artery and only once on the posterior wall. The anterior position was also encountered in the unruptured case. This gives a ratio of 3.1 of anterior to posterior aneurysms.

9. Direction and Extension of the Aneurysm.

In all four cases aneurysms of the thoracic aorta were found to extend to the right. This suggests a prevalence of extension in that direction.

10. Probable Mechanism of Formation of Aneurysm.

On a mechanical basis, the thrust of the column

of /

of blood with each systole of the heart is presumably greater on the wall of the ascending portion of the arch than on its distal parts. The combination of the degenerative process in the media of the artery and the force of the impact of the arterial thrust on the weakened wall of the vessel, greatly favours the formation of the aneurysm in the first portion of the ascending aorta than in other parts distal to this site.

11. Fatal Symptoms.

In case I. no such symptoms developed, although preceding the fatal issue, breathlessness occurred on account of exertion. In case II. death ushered in with collapse while at rest and no symptoms, apart from those of collapse, occurred. In case III. agonising pain in the chest immediately preceded death.

Therefore the only symptom encountered with rupture of an aneurysm of the thoracic aorta was agonising pain in the chest in one third of the cases. Effort was undoubtedly a factor in this case.

12. Cause and Mode of Death.

The immediate cause of death in these cases was cardiac tamponade resulting from rupture of the aneurysm into the pericardial sac. Death was almost instantaneous in two cases, and in one case occurred in a few minutes.

13. Incidence of Rupture of Aortic Aneurysms.

Of the four cases of aneurysms of the thoracic aorta, three had ruptured, an incidence of 75%. Incidence of rupture of aneurysms of the abdominal aorta was 50%. The ratio of rupture in these two groups was 1.5 : 1 respectively.

14. Predisposing Causes of Rupture.

Physical exertion was encountered in two cases out of three, and was rather of a severe degree in one case. In the third case, no such factor was present and, indeed, death occurred while the patient was at rest.

15. Past History.

All patients presented symptoms of a varying duration between 6 months and three years. These were, breathlessness on exertion, moderately severe pains in the back and an unproductive cough. However, these symptoms did not interfere with the activities of the patient, and all the patients were engaged in work up to time of death. There were no other pathological findings, apart from the aneurysm of the aorta, to account for the symptoms.

C O M P A R A T I V E S T U D Y

1. Incidence of Aneurysms and Site.

Aneurysms /

1. Incidence of Aneurysms and Site.

Aneurysms of the thoracic aorta seem to form a small proportion as a disease entity. There were in all over 5,000 cases of thoracic aneurysms reported in literature (Boyd 1924). This observation is in accordance with the incidence of such aneurysms in the series under study. In agreement, Kampmeier (1938), stated that the incidence of this condition has decreased by more than half in the past decade. Contrary to this statement, Boyd (1924) is of the opinion that thoracic aneurysms are not rare and gives a mortality from them in American cities as between 0.1% - 0.5%.

2. Sites of Aneurysm.

The general opinion is that the ascending portion of the aorta is the most frequent site of aneurysms (Boyd 1924), (Dressler 1942), and (Lucke and Rea 1923). The present series reflects this feature. Boyd gave the ratio of the incidence of aneurysms in the various portions of the aorta as approximately 10 : 7 : 3 : 1, for the ascending, transverse and descending segments of the aortic arch, and the descending thoracic aorta respectively.

Lucke and Rea in their series of 321 aortic aneurysms found 173 in the arch of the aorta, 40 in the abdominal aorta and 31 in the thoracic aorta.

Lowenberg /

Lowenberg and Baer (1946) referred to Kampmeier, and Levitt and Levy to have given the following figures respectively for aneurysm of the descending thoracic aorta: 4.7% and 4.3%. He also mentioned that Brindly and Schwab have found that 2% of aortic aneurysms were in the lower thoracic aorta. He emphasised that the location of these aneurysms renders them frequently undiagnosed during life, giving an example of a case in a male, aged 61, complaining of abdominal pain which was aggravated by cough and deep breathing, who died suddenly in a paroxysm of cough with profuse haemoptysis from rupture of a thoracic aneurysm into the left lung. The only presenting symptom in some cases may be an intractable hiccough (Lowenberg and March, 1937). It is significant, therefore, that in aneurysm of the descending aorta the process may be latent and the signs so negligible that recognition may tax the ability of experienced physicians. In this connection Osler noted, "There is no disease more conducive to clinical humility than aneurysm of the aorta".

3. Dynamic Dilatation of Thoracic Aorta Simulating Aneurysm.

Not infrequently a dynamic dilatation of the thoracic aorta is confused with an aneurysm. The recognition of such a condition is of the utmost importance. Morgagni was acquainted with this condition /

condition and described its occurrence in three conditions: aortic insufficiency, neurotic states, and anaemia (Osler 1927). Bayley and Mich (1933) defined the condition as an increase in the size of the lumen of the aorta for a variable distance along its course, without structural disease of the aortic wall. In 1886 Hare reported a most instructive case as an aneurysm of the thoracic aorta in a girl, aged 18 years, with pain in the left side of the chest and left arm. He described, "An egg-shaped protrusion in the suprasternal notch, very expansile and bulging at each systole of the heart. Over the protrusion was a thrill and bruit". Osler, who saw the patient during life, stated that at autopsy a few months later, he was not surprised at finding the lumen of the aorta too small to admit the index finger.

Sheldon observed the condition in a boy, 10½ years old, who suffered from chronic nephritis with hypertension and secondary anaemia, and an enlargement of the aortic arch radiologically. At autopsy, one month later, the aortic arch was found of a normal size. Sheldon cited a similar case described by Evans.

Macree (1910), Brown (1912) and Lankford (1925) reported permanent enlargement of the cavity of the aorta due to syphilis and atherosclerosis. This condition most commonly involves the ascending aorta.

It was described by Hodgson in 1815, and is referred to as the *Maladie de Hodgson* by the French. Bayeley described three cases, one in a female, aged 35, another in a boy, aged 17 and a third in a boy aged 14.

4. Direction and Extension of the Aneurysm.

As regards tendency of aneurysm of the ascending portion of the aorta to develop towards the right side, as was found in the series under study, Dressler (1942) is of the opinion that distention and displacement of the aortic wall in this direction are favoured by the minimum resistance offered by the lungs, as well as by the fact that the right wall of the ascending aorta receives the greatest impact from the column of blood which is expelled during systole; for the outflow tract of the left ventricle runs an oblique course from low on the left to high on the right. He referred to those extending towards the left side of the chest as being exceedingly rare, and create unusual clinical pictures, which may cause considerable clinical difficulties.

Lucke and Rea (1923) on the other hand, found the aortic extension posteriorly and to the left, and that the oesophagus was displaced posteriorly and to the right. They also stated that the only aortic aneurysm that can displace the oesophagus anteriorly and to the left is an aneurysm at the hiatus. In

this /

this connection, Roesler (1943) and Shanks et al (1938) have pointed out that this deviation of the oesophagus may be produced by one another rare aortic abnormality; namely a right-sided aortic arch.

5. Age Incidence.

Boyd (1924), from a study of 4,000 cases from literature, states of age incidence, that it rises slowly from birth and reaches maximum in the period from 36 to 40 years and then falls more slowly, but the condition may occur at any age. He speaks of the curve of incidence in males as rising slowly from birth until the 25th year and then rapidly reaches a maximum in 36 to 40 years, and that the curve of descent is more gradual. The course of the curve confirms the universally accepted dictum that the maximum frequency of aneurysm corresponds to the period of greatest incidence of syphilitic vascular disease. The average age incidence, as suggested in the series under discussion, is in accordance with maximum age incidence of 40 years. In females the largest number of aneurysms is found in the 46 to 50 year period, ten years later than the maximum for males. Up to 41 - 45 year period, the curves are similar, but in the next five years, instead of the expected fall, there is a rise. From this age until 65 there is a steady fall. At 75, the number
of /

of cases for both sexes is equal.

6. Sex Incidence.

All statistics are in agreement that a marked predominance occurs in males although figures differ from one to another.

Boyd (1924) gives the incidence of thoracic aneurysms as 5 - 6 times as common in males as in females. White (1946) gives a ratio of 10 to 1, and Boyd referred to other statistics with a ratio from 10 to 3 up to 11 to 1. Osler gives the incidence in males to females as 5 to 1. The preponderance of the male sex is attributed to two causes, (a) syphilis and (b) trauma (Boyd), as of a more frequent occurrence in this sex.

7. Race and Occupation.

Observers note that coloured races are notoriously liable to aneurysm, (Osler, White and Boyd). White gives an incidence of 6 to 7 times as common in black as in white people. This is attributed to the following causes:-

- (i) Greater incidence of syphilis in negroes.
- (ii) Less satisfactory treatment among negroes.
- (iii) Heavy type of negro labour.

Osler stated that in India aneurysms are rare, though syphilis and arterial disease are common. He

also /

also noted that the disease was more common in Great Britain than on the Continent. In contrast he found that among 19,000 postmortems at Vienna there were 230 cases of aneurysms, while among 18,678 post-mortems at Guy's Hospital there were 325 cases.

No such factor was noticed in this series.

8. Determining Causes.

(a) Syphilis stands out beyond any other cause as emphasised by all observers. White (1946) gives an incidence of 90%. Wilburne and Taylor (1942) give an incidence of 85%. However, Martland (1930) gives an incidence of 38%.

(b) Atheroma and rarely trauma account for 15% (Wilburne and Taylor 1942).

In present series syphilis accounted for all aneurysms of the ascending portion of the aorta.

(c) Trauma, tuberculosis and mycotic infection play a minor role (Boris 1942).

Ruffin et al (1941), in an analysis of 9,600 autopsy records found:-

Syphilitic aneurysms of the thoracic	
aorta.....	60 cases
Syphilitic aneurysms of the abdominal	
aorta.....	3 cases
Atherosclerotic aneurysms of the	
abdominal aorta.....	27 cases
Atherosclerotic aneurysms of the	
thoracic aorta.....	3 cases

The ratio of syphilitic aneurysms of the thoracic aorta to atherosclerotic aneurysms of the abdominal /

abdominal aorta is almost the same in this series as well as in the series under discussion. However, no syphilitic aneurysms were encountered in the abdominal aorta in present series.

Congenital syphilis has been blamed on many occasions to have caused aneurysmal dilatation.

Wilson and Marcy appear to have been the first to verify by necropsy the existence of an aortic aneurysm caused by congenital syphilis.

Escudero (1912) reported on two congenital syphilitic brothers 4 and 6 years of age, who had a cylindrical dilatation of the thoracic arch, together with aortic stenosis.

Acuna (1915) and Heiman (1919) presented cases of aneurysms of the thoracic aorta in children, 12, 13 and 14 years of age, with congenital syphilis and positive blood Wassermann reactions.

Acuna, Winocu and Orosco verified in a congenitally syphilitic girl, 6 years of age, the existence of multiple aneurysms of the aortic arch and thoracic abdominal aorta. They also mentioned the discovery by Maniogu of an aneurysm in a boy 8 days old.

Mycotic aneurysms resulting from gonococcal infection, according to Currens and Faulkner (1943), have been encountered in 9 cases reported in literature. Five of these cases occurred in males between the ages of 12 to 28, and four in females between the

ages /

ages of 23 and 38. However, they added the tenth case of a male, aged 41, who had a history of syphilis and gonorrhoea 25 years previously. The patient suffered from attacks of cardiac asthma over one year before his death, and his fatal issue was determined suddenly by an attack of acute pulmonary oedema. His blood culture was positive for gonococci. No such case, to their knowledge, had ever been reported.

The interesting observation in these cases is the comparatively young age of the patient. All were under 30, with the exception of two, one aged 38, and the other 41 years.

9. Multiple Aneurysms

These were reported by Kampmeier (1935) who in a study of 270 cases of aneurysms of the thoracic aorta found 9 cases exhibiting multiple lesions, an incidence of 3.3%. In another series he reported an incidence of 3.6%. An incidence of 8% was reported by Colt (1927), and 21% by Lucke and Rea (1923).

Three cases were given by Wilburne, all syphilitic and in males of 36, 63 and 79 years of age. The second case presented four saccular aneurysms, while in the other two each presented three saccular dilatations. In spite of the lesions in these cases, symptoms /

symptoms did not appear until relatively late in life.

Ruffin et al (1941) reviewing the literature found only one reported case of multiple saccular aneurysm of the aorta of a non-syphilitic origin.

10 Mechanism of Formation of Aneurysm.

Martland in discussing this mechanism stated that the weakened arterial wall by syphilitic affection stretches under the shock of systolic filling. The ascending portion of the aorta becomes dilated and may pouch out. He also noted that if an aortic reflux develops, it may relieve the strain and that aneurysms are less likely to develop if reflux has developed early.

11. Time Interval between Infection and Production of First Symptoms of Aneurysm.

Boyd (1924), in a study of 200 cases gave an average interval of 20 years. The minimum time was one year, though syphilitic aortitis has been reported within six months. The upper limit was 56 years after infection. He concludes that these figures, outside of showing the usual time interval, emphasise that a syphilitic individual remains throughout potentially aneurysmal.

12. Longevity.

Stewart and Garland (1932), reported on a case of a remarkable example of prolonged survival and

continued /

continued good health over a period of 29 years in a male, who was in full work as a janitor. Autopsy revealed a thickened, partly calcified aneurysm of the aortic arch.

However, the course of the malady may be rapid and fatal as in a case reported by Shärer and Pietrafesa (1944), of a girl 18 years old, who died only three months after her first symptoms.

In the cases under study no history or clinical evidence was available to assess the period of longevity, but the age of the patient in case III. suggests a duration of longevity of some years standing.

13. Rupture.

Rupture is the most dreaded event. It usually occurs into the pericardial sac or the pleural cavity. Unusual ruptures occur into the oesophagus (Boris 1942). Irwin and Frankel (1945) described an unusual case of syphilitic aortic aneurysm in a man aged 60, which was found to have extended along the whole course of the aortic arch and the descending thoracic aorta down to the first two inches of the abdominal aorta. The perforation occurred into the posterior wall of the stomach.

Other recognised uncommon sites of rupture are the auricles, great veins, and pulmonary artery.

14. Interval between Rupture and Death./

14. Interval between Rupture and Death.

Death is known either to occur suddenly or after a short period from rupture, but there are always exceptions. A survival period of seven days after a rupture was reported by McFarlane, (1932) in a case of a man aged 55, whose aneurysm was in the abdominal aorta. This case is similar to the one referred to in connection with the present series, only that survival period after rupture did not exceed three days.

The apparent observation from these findings is that ruptures of aneurysms of the abdominal aorta are not likely to be immediately fatal when compared with those of the thoracic aorta.

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REPORT

1. General Remarks

The purpose of this report is to provide a summary of the results of the investigation conducted by the author. The investigation was carried out in accordance with the instructions of the Ministry of Defense of the Soviet Union. The results of the investigation are presented in the following sections.

(a) Data from the investigation are presented in the following table:

(b) The results of the investigation are presented in the following table:

(c) The results of the investigation are presented in the following table:

SECTION III

CONCLUSIONS

The results of the investigation are presented in the following table:

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C O N C L U S I O N S

I. General Statement:

The review of my own 3,250 case-records of necropsies for the Medico-Legal Department of the Ministry of Justice of Egypt over a period of nine years has revealed the following data:-

- (a) Death from natural causes occurred in 1,980 cases; 60.9%.
- (b) Sudden death occurred in 760 cases; 38.4% of those dying from natural causes.
- (c) Sudden cardiac death occurred in 365 cases; 48% of the cases of sudden death.

The records of sudden cardiac death (365 cases) were extracted for study.

These cases were classified and grouped on a pathological basis.

The study of these cases was undertaken from consideration of the following points:-

1. Incidence of the disease in post mortem series.
2. Age incidence.
3. Sex incidence.
4. Race.
5. Bodily constitution.
6. Past history.
7. Social conditions.

8. Associated diseases.
9. Predisposing factors.
10. Symptoms prior to death, and their duration.
11. Duration of life after the onset of the disease.

II. The main groups of diseases resulting in sudden cardiac death in this series were as follows:-

- (a) Coronary artery disease and its sequelae.....83.1%
- (b) Infectious process directly affecting the myocardium..... 7.6%
- (c) Syphilitic affection of the aorta, aortic valve, and coronary arteries..... 4.6%
- (d) Rheumatic fever affecting the heart and aortic valve..... 3.0%
- (e) Adiposity of the heart..... 1.7%

III. Incidence of sudden cardiac death predominated in males over females in a ratio of 2.7 : 1.

IV. A summary will now be presented of the causes of sudden cardiac death in this post mortem series on a basis of pathological anatomy.

CORONARY ARTERY DISEASE

Coronary artery disease was responsible for the vast majority of sudden cardiac deaths.

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The following pathological entities were classified:-

- | | | |
|-------|---------------------------|---|
| (i) | Coronary atherosclerosis | : 212 cases; representing 58% of the series. |
| (ii) | Coronary Occlusion | : 85 cases; representing 23.1% of the series. |
| (iii) | Coronary ostial occlusion | : 3 cases; representing 0.9% of the series. |

CORONARY ATHEROSCLEROSIS

Coronary sclerosis is the most frequent disease of the coronary arteries.

In all cases under study, a severe involvement of the coronary arteries by atherosclerosis was encountered, and death was considered as being due to coronary disease alone.

The cases were further divided, on a pathological basis into two groups in respect of the size of the heart:-

- (i) Those with cardiac hypertrophy.
- (ii) Those without cardiac hypertrophy.

Cardiac hypertrophy was considered indicative of a coexisting arterial hypertension.

The criteria used in classifying the hypertensive group were threefold:-

- (a) Evidence of cardiac hypertrophy as stated above.
- (b) Absence of valvular lesions which may account for cardiac hypertrophy.

(c) /

- (c) Absence of any other cause of cardiac hypertrophy.

The non-hypertensive group was found to predominate slightly over the hypertensive group in a ratio of 1.2 : 1.

A comparative study of these two groups in respect of age and sex incidence revealed the following:-

- (a) In both sexes, the span of life was longer in the non-hypertensive group than in the hypertensive group, and was especially so in the male sex.
- (b) In both groups the span of life was shorter in the female than in the male sex.

The evidence suggests that prognosis of coronary atherosclerosis in the female sex is less favourable than in the male sex, irrespective of the presence or absence of hypertension.

- (c) The highest incidence of deaths occurred at the age of 50 years (45.8%), and the second highest incidence at the age of 45 years (18.9%), irrespective of the presence or absence of hypertension.

The evidence suggests that coronary sclerosis is essentially a disease of middle life.

- (d) In the hypertensive group the rise of deaths to the peak was sharper than in the non-hypertensive group, and the fall from the peak was sudden in both groups, irrespective of sex difference.

The evidence suggests that hypertension in coronary sclerosis is of a less favourable prognosis in the first fifty years of life.

Coronary atherosclerosis in general is a disease
of /

of the male sex, males predominating over females in a ratio of 3.1 : 1. This predominance in the male sex was mainly noted in the non-hypertensive group (4.7 : 1), and was found to have decreased in the hypertensive group (2:1 only).

The evidence suggests that females are more afflicted with hypertension than males.

Heredity appeared to be a definite aetiological factor in this disease, (62.8%).

Obesity was also rated an aetiological factor in coronary atherosclerosis and was especially noted in the hypertensive group (40.8%).

The evidence suggests that obesity overtaxes the ischaemic heart.

Diabetes mellitus was an associated disease in 14.1% of the cases.

Smoking and alcohol were not significant factors.

A seasonal incidence was disclosed in the present series; 58.8% of the cases occurring in the winter and autumn.

The evidence suggests that cold probably acts as a vasoconstrictor or at least prevents vasodilatation of the coronary arteries.

With regard to the effect of cold weather in precipitating anginal attacks, the occurrence of the disease in 18 carters, 10 cabmen, and 8 farmers; an incidence of 16.9% is supporting evidence. But

the /

the equivalent incidence noted among the professional classes (16.9%) casts some doubt on the significance of the effect of cold weather in predisposing to anginal attacks.

In respect of symptoms prior to death, two groups of patients were encountered; one group presenting symptoms (62.3%), and the other dying without showing symptoms (22.6%).

As coronary atherosclerosis in the second group of patients was of a more marked degree than in the first group, the evidence suggests that a fibrosed myocardium is less sensitive to ischaemia, and therefore, patients are less prone to show symptoms prior to death.

In respect of the degree of coronary sclerosis it was also found that:-

- (a) The number of anginal attacks during life was proportional to the degree of sclerosis.
- (b) The duration of life after the development of the attacks was inversely proportional to the degree of sclerosis.

The evidence suggests that the span of life is shorter in patients with a severe degree of coronary sclerosis.

Physical effort was a significant predisposing factor to fatal attacks in a large proportion of cases in the first group (60.7%). In the second group effort was a less significant factor (37.5%).

The average duration of life after the development of

the /

the attacks was three years and eight months in 45.3% of the cases.

In respect of the incidence of sclerosis in the main arteries, it was found that the left coronary artery was involved 1.4 times as often as the right artery.

Instantaneous deaths occurred in 4.2% of the cases.

Very sudden deaths were more likely to occur in the non-hypertensive group than in the hypertensive group (2 : 1 respectively.).

The evidence of fibrosis of the myocardium in these cases suggests a previous state of myocardial ischaemia due to reduction of the coronary blood flow.

It is, therefore, suggested that an irrecoverable state of myocardial ischaemia is the cause of death.

CORONARY OCCLUSION

The records of cases of coronary artery occlusion studied post mortem revealed the following:-

Coronary artery occlusion is an incident in atherosclerosis of the coronary arteries; atherosclerosis being the basis of all such occlusions.

Occlusions were either caused by intramural haemorrhages or thromboses; the former were encountered 2.1 times as often as the latter. It is, therefore, suggested that the ultimate differentiation between

these /

these two types of occlusions is entirely a histological interpretation.

The degenerative process consequent upon the development of atheromatous plaques in the coronary arteries with the resultant thickening of the intima is followed in the majority of cases by new capillary formation in relation to these plaques. The fragility of the newly formed capillaries is attended with rupture resulting in the formation of a haematoma and subsequent coronary occlusion.

In most cases extension of the haematoma by rupture through the intima is followed by thrombosis.

The left coronary artery was the vessel more frequently occluded than the right in the ratio of 1.8 : 1, and the anterior descending branch of the left coronary artery was the branch most frequently involved.

Evidence of collateral circulation occurring subsequent to old occlusions of the anterior descending branch of the left coronary artery was found in a small proportion of cases (8.2%). This was shown by the occurrence of apical anterior infarctions when this branch was severely occluded by old thrombosis.

The sex incidence of the disease follows essentially that of coronary atherosclerosis; males predominate over females in the ratio of 2.4 : 1.

In /

In respect of the age incidence in both sexes it was found that:-

- (a) The disease occurred earlier in life in males than in females, the youngest male being 29 and the youngest female 40. It also occurred later in life in males than in females; the oldest male being 70 and the oldest female 65.
- (b) The major incidence of deaths in males occurred 5 years later in life than in females.
- (c) In males the rise to the peak was in a stair-like fashion, while in females it was very sudden, and the peak was higher than in males.

The graph, Fig 6. suggests that the disease is generally milder in males than in females, and that an aetiological relationship between such age incidence in the female sex and the menopause may exist.

Obesity was a significant factor especially in the female sex (36% in females and 26.7% in males).

Heredity appeared of almost equal significance (22.4%).

Hypertension was rated an aetiological factor in the ratio of 28.3% in males and 32% in females.

Diabetes mellitus was associated with coronary occlusion in 11.8% of the cases.

The disease seems to occur more frequently among the labouring class than among the professional classes.

Smoking and alcohol were insignificant factors

in /

in predisposing to the disease.

The majority of deaths occurred in winter and autumn, 42.4% and 25.8% respectively, which suggests that cold weather may be a predisposing factor.

Symptoms prior to death were precordial pain, dyspnoea, epigastric pain, nausea and vomiting.

The duration of symptoms varied from one-half hour to six hours.

Ordinary physical activity preceded fatal symptoms in a large proportion of cases (62.4%), while deaths occurred during sleep in 30.6% of the cases.

The evidence suggests that physical activity is decidedly an aetiological factor predisposing to the attacks in contradiction to the commonly held view that such attacks are more likely to occur at rest.

Mortality rate in the first attacks was 17.6% and the average duration of life after an episode of coronary occlusion was 1.4 years.

Pulmonary embolism as a cause of sudden death was encountered in 8.2% of the cases.

CORONARY OSTIAL OCCLUSION

Three cases of syphilitic occlusion of the coronary orifices, two males and one female, were reported upon and analysed with cases from the literature.

The /

The cases represented 0.9% of the series.

Death occurred at ages of 43, 40, and 35 years; the youngest patient was a female (Sudaneze).

The coronary arteries were involved only at their ostia and not in their length.

The right coronary orifice was more frequently occluded than the left in a ratio of 2 : 1.

The heart was moderately hypertrophied in all cases. No evidence of syphilitic myocarditis was encountered in any case.

Myocardial infarction did not occur with occlusion of either the right or left coronary artery. This suggests that narrowing or atresia of the coronary orifices takes a long period of time in its development, and presumably a collateral circulation was established.

Death in all cases was sudden, occurring within 2 to 5 hours of fatal symptoms.

Symptoms prior to death were cardiac pain, dyspnoea, cough, and vomiting.

A slight rise of temperature was noted in all cases.

SPONTANEOUS CARDIAC RUPTURE

Spontaneous cardiac rupture occurred in 9 cases accounting for 2.6% of sudden cardiac deaths.

The /

The recognised causes of rupture were classified as follows:-

- (i) Rupture due to acute myocardial infarction: 2 cases representing 0.6% of the series.
- (ii) Rupture of cardiac aneurysm due to old standing coronary thrombosis: 5 cases representing 1.4% of the series.
- (iii) Rupture due to adiposity of the cardiac muscle: 2 cases representing 0.6% of the series.

RUPTURE DUE TO ACUTE MYOCARDIAL INFARCTION

Two cases of sudden death due to rupture of the heart following acute myocardial infarction were described in males of 60 and 65 years of age.

In both cases the infarction involved the left ventricle and was due to thrombotic occlusion of the anterior descending branch of the left coronary artery, and the rupture appeared to be precipitated by exertion taking place soon after the infarction had occurred.

No symptoms seem to have attended the fatal issue, and in both cases death was due to acute cardiac tamponade from haemopericardium.

Death occurred within fifteen minutes of rupture.

The cases were correlated with others reported

in /

in the literature with respect to:-

- (a) Age incidence
- (b) Sex incidence
- (c) Racial factor
- (d) Aetiology
- (e) Pathology

RUPTURE OF CARDIAC ANEURYSM
DUE TO
OLD STANDING CORONARY OCCLUSION

The age and sex incidence of these cases suggest an association between development of cardiac aneurysm and coronary artery disease.

The site of aneurysms, in the anterior wall of the left ventricle involving the apex of the heart, and the absence of aneurysm of the auricles, further suggest the relationship between cardiac aneurysm and coronary occlusion.

Coronary occlusion is much more common in the anterior descending branch of the left coronary than in the right coronary artery or its branches.

The cause of cardiac aneurysms in this series was a healed myocardial infarction resulting from coronary thrombosis of some standing.

The presence of cardiac aneurysm is not usually associated with any symptoms due specifically to the aneurysm /

aneurysm, and the condition may be missed during life unless a radiological examination is made.

Cardiac hypertrophy in all cases, the presence of arteriosclerosis of the kidney in two cases, and the previous health of the patients suggest that hypertension has been present in these cases.

Rupture of cardiac aneurysm may occur at any time, and this may be of importance in medico-legal work, although it must be realised that a scarred cardiac muscle, especially when in a state of aneurysmal dilatation, is always in imminent danger of rupture at any time.

The immediate cause of death in these cases was cardiac tamponade from haemopericardium.

The incidence of this condition in the labouring class may be explained by the fact that this class of patient cannot afford to remain in bed long enough to recover from the effects of coronary artery disease. They invariably resume work as soon as their main symptoms have ceased. Thus, this factor predisposes to weakness of the infarcted cardiac muscle and subsequent aneurysmal dilatation.

ADIPOSITY OF THE HEART

There seems to be no doubt that cardiac adiposity
is /

is a cause of sudden death (6 cases).

Death may be due to:-

- (i) Acute cardiac failure (4 cases);
representing 1.1% of the series.
- (ii) Rupture of the heart with haemopericardium and cardiac tamponade (2 cases);
representing about 0.6% of the series.

Both of these episodes are due ultimately to replacement by fat of the heart muscle fibres.

Replacement by fat involves mainly the right ventricle.

Cardiac adiposity occurs in genuine adiposity and is due to the same factors.

Chronic alcoholism was an outstanding aetiological factor in five of the six cases.

Severe physical exertion preceded cardiac rupture in the two cases observed.

Death due to acute cardiac failure may occur even at rest.

Precordial pain and dyspnoea may usher in the fatal issue.

M Y O C A R D I T I S

Thirty-two cases of myocarditis due to different infective aetiological factors were discussed.

The aetiological factors encountered were:-

- (i) Influenza
- (ii) Diphtheria

(iii) /

- (iii) Rheumatic fever
- (iv) Pulmonary tuberculosis
- (v) Typhoid fever
- (vi) Malaria
- (vii) Relapsing fever
- (viii) Dysentery
- (ix) Measles.

The diagnosis is necessarily a pathological one since there is no uniform clinical picture.

The constant pathological findings in all cases were cardiac dilatation and flabbiness of the heart. Cloudy swelling and fatty degeneration were relative in their development to the time incidence of the original illness.

Sudden death from this condition represents 8.7% of the series. It may occur in some cases as late as several months after the onset of the original illness.

VALVULAR DISEASES

Sudden cardiac death due to chronic affections of the aortic valve occurred in 4.7% of the series.

Three aetiological factors were disclosed:

1. Syphilitic aortic disease resulting in aortic incompetence; 10 cases (58.8%).

2. Rheumatic aortic disease resulting in aortic incompetence; 3 cases (17.7%).
3. Calcareous aortic disease resulting in aortic stenosis; 4 cases (23.5%).

Syphilitic Aortic Valvular Disease:

Syphilis is primarily a disease of the aorta. Incompetence of the valve was due to widening of the commissures by extension of the syphilitic process between them, although in all cases the aortic ring was also dilated.

Cardiac hypertrophy and dilatation of the left ventricle were also encountered in all cases.

In none of the cases was there any involvement of the coronary vessels by the syphilitic process.

Sudden death occurred in 10 out of 26 cases of syphilitic aortic incompetence studied post mortem; an incidence of 38.5%. These 10 cases represent an incidence of 2.8% in the whole post mortem series of sudden cardiac death.

Age incidence at death was 44.8 years, (average).

Males predominate over females in the ratio of 4 : 1.

The interval between the primary infection and death was 22 years (average in three cases).

Fatal symptoms occurred in 80% of the cases. Dyspnoea was the main symptom. Giddiness, palpitation and precordial pain were infrequent.

Physical /

Physical exertion predisposed to development of fatal symptoms in one half of the cases and the duration of the symptoms was from one half to two hours.

Symptomless death occurred in 20% of the cases. It occurred with dramatic suddenness, and was not related to any form of physical exertion.

Rheumatic Aortic Valvular Disease:

Rheumatic vegetations show a predilection to localise on the ventricular aspect of the cusps of the aortic valve.

Sudden death occurred in 3 cases; representing 0.8% of the series.

Rheumatic affections of the aortic valve predominate in males over females in the ratio of 2:1.

Death occurred at the fifth decade of life.

Fatal symptoms; palpitation and dyspnoea occurred in one case only, and were not related to any form of abnormal physical exertion.

Calcareous Aortic Disease:

Calcareous disease of the aortic valve was encountered in 4 cases; representing 1.1% of the series.

The disease showed a predilection to occur in the male sex; all cases being males.

Deaths occurred mainly from the fifth decade

onwards /

onwards and, therefore, a long span of life may be expected with the condition.

Fatal symptoms; dyspnoea, palpitation, and precordial pain occurred in three cases while at work. The fourth case was found dead on the road.

The stenotic lesions reduced the orifice of the aortic valve to from 5 to 10mm. in diameter, and a moderate degree of cardiac hypertrophy was found.

The aorta presented a severe degree of atherosclerosis, and the coronary arteries were sclerotic.

In none of the cases was the mitral valve involved by the calcareous process.

RUPTURE OF ANEURYSM OF THE AORTA

Six cases of aneurysm of the aorta were described.

In four of these cases the aneurysms involved the ascending portion of the aortic arch, and in the remaining two cases the abdominal aorta was involved.

Syphilis was the cause of all aneurysms involving the ascending thoracic aorta; the abdominal aortic aneurysms were atherosclerotic in origin.

All cases occurred in males.

In aneurysms of the ascending aorta, rupture into the pericardial sac occurred in three cases, and death was almost immediate from cardiac tamponade.

Rupture /

Rupture occurred in one of the two cases of abdominal aneurysm, and death was delayed for three days.

Exertion appeared to be a factor causing rupture in two of the thoracic aneurysms.

In all cases of aneurysm of the thoracic aorta, the victims were unaware of their disease, since no definite symptoms were present.

The aneurysms of the ascending aorta were found anteriorly more commonly than posteriorly (3 : 1).

Extensions of the aneurysm to the right was noted in all the ascending aortic aneurysms.

Sudden death due to rupture of thoracic aneurysm represents 0.9% of the series.

The present series of cases of thoracic aneurysm were correlated with other larger series in the literature with respect to

- (i) Aetiology
- (ii) Age incidence
- (iii) Clinical features
- (iv) Pathology.

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